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THE CHEMISTRY OF THE BLOOD IN DISEASES OF THE SKIN

A STUDY OF EIGHT HUNDRED AND SEVENTY-FIVE CASES *

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Perhaps the most fallible link in the knowledge of diseases of the skin is their causation. Many dermatoses are cured without our being able to ascertain the cause of the condition for which we have given treatment. Such results are perhaps gratifying from the patient's standpoint but not from our own, as it is highly unsatisfactory to treat a patient for a disease without knowing its cause.

The skin is more exposed to external insults—mechanical, chemical and parasitic—than any other organ. It is perhaps natural that there should exist a considerable diversity of opinion among dermatologists as to the dominance of external versus internal factors in the causation of many diseases of the skin, particularly the inflammatory dermatoses.

The controversy on eczema itself covers a considerable literature. The skin plays a prominent part in immunologic processes in the body, and many so-called eczemas are believed to be of allergic origin. Eczema is, in fact, less a disease than a form of cutaneous reaction due to external, internal and combined causes. It cannot clinically or histologically be distinguished from dermatitis.

For the past seven years we have been studying the chemistry of the blood in diseases of the skin. All of the patients studied were private patients, and they could be followed more persistently than clinical cases. While considerably more than 1,000 cases were studied, only 875 reports were sufficiently complete to warrant inclusion in this publication. The blood was always taken in the forenoon within from one to four hours after breakfast. On account of the time relationship to breakfast the figures for dextrose were not always accurate. The examinations were all made by the same chemist or under his imme-

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diate supervision and in his presence. The only tests included in this report are for nonprotein nitrogen, urea nitrogen, dextrose and uric acid.

INFLUENCE OF SEX AND OF AGE

Sex.—One of the most evident deductions from our figures is that with respect to the nitrogenous constituents of the blood, age and sex play a distinct rôle. So striking is this that it is perhaps unscientific to inquire what is the maximum normal of nonprotein nitrogen, urea nitrogen or uric acid in the blood, but rather what is normal for a male or a female subject of a given age. We have long had this impression, and a careful study of the figures definitely confirms it. This is well shown in chart 1.

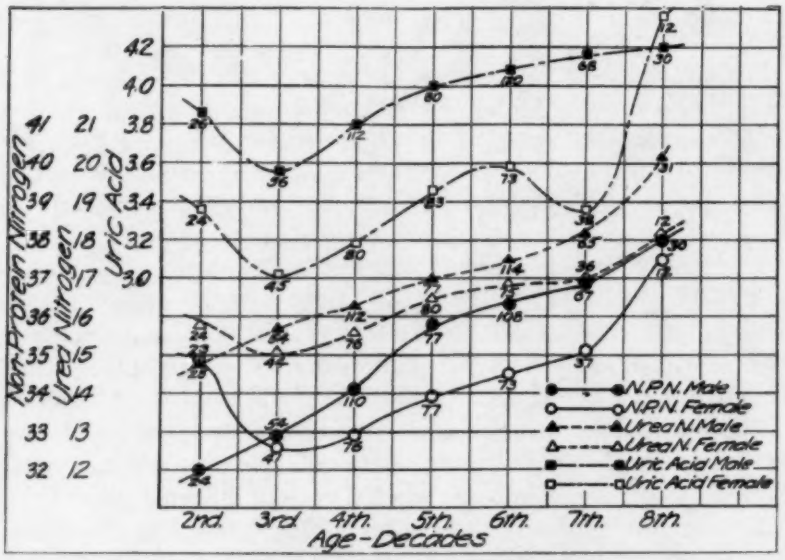

Chart 1.—Amount of nonprotein nitrogen, urea nitrogen and uric acid in the blood, according to sex and age. In this and the following chart, the figures on the vertical scales indicate milligrams of the respective substances.

The discrepancy between the sexes is more pronounced with respect to uric acid. Generally speaking, men between the age of 20 and 70 have on the average about 0.6 mg. more uric acid per hundred cubic centimeters of blood than women. There are perhaps a number of causes for this difference. One factor may be that, generally men eat more and partake of more purin-containing foods than women. In a general way, it may also be stated that the men studied by us had almost 1 mg. more of urea nitrogen per hundred cubic centimeters of blood than the women. Nonprotein nitrogen also showed parallel differences.

Age.—A study of the records of our patients shows a steady rise in uric acid, urea nitrogen and nonprotein nitrogen from the third to the eighth decade of life. Whereas at the age of 30 the average uric

acid for men is about 3.5 mg., it is 4.2 mg. at the age of 80. Curiously, in the age period from 10 to 20 the figures of the nitrogenous constituents of the blood, and particularly for uric acid, are higher than those at the age of 30. We have no explanation for this curious observation.

The urea nitrogen for males at the age of 30 was on the average between 15 and 16 mg., whereas at 80 it was over 20 mg., the rise being gradual between these periods. Nonprotein nitrogen rose from 33 mg. at the age of 30 to 38 mg. at the age of 80.

These figures are of interest and suggest that as persons increase in age there is in general an enhanced renal dysfunction. Old kidneys apparently do not function as well as young ones.

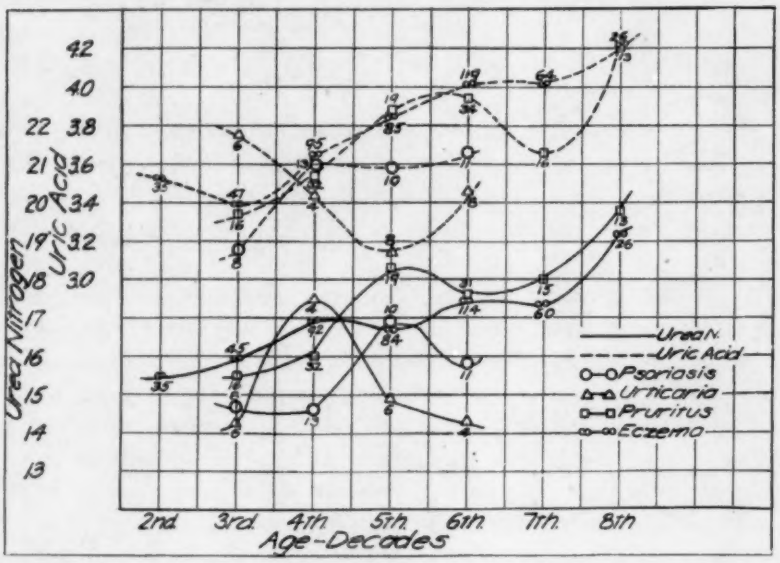

Chart 2.—Amount of urea nitrogen and uric acid in the blood in relation to age and the cutaneous disease present.

Of the 875 cases studied of special diseases of the skin, 455 were designated as eczema. It is obvious that this group contains cases that were due to many and varied causes. After all, eczema is no longer regarded as a disease but rather as a dermic reaction. There is one school that considers external irritants as the dominant cause of eczema or dermatitis, whereas another believes that systemic conditions play a large rôle. To be sure, both internal and external factors may operate in the production and continuation of such conditions.

Allergy, gout, diabetes, intestinal toxemia, endocrine disturbances, renal disease and many other causal conditions, including a great variety of external irritants, may play a determining part.

Our reports on blood chemistry have been studied with a view to throwing some light on this complex subject. We have had no preconceived theories to prove. We have approached the subject from an objective point of view for the purpose of ascertaining the facts, and we shall present them for what they are worth.

ECZEMA

We have been impressed with the comparatively small part which hyperglycemia plays in the production of cutaneous diseases. While we have seen a number of cases of eczema in which the condition was associated with diabetes and was kept up by this condition, the number of instances in which eczema was caused by a pathologic increase of dextrose in the blood was small. Moderate increases in blood dextrose were often due to the fact that the blood was taken too soon after breakfast. On reexamination after fasting, the blood was usually normal.

The case was entirely different, however, with regard to the nitrogenous constituents of the blood. An excess of nonprotein nitrogen, urea nitrogen and uric acid was distinctly more common than in other dermatoses, with the exception of pruritus, especially generalized pruritus.

We called attention, several years ago, to hyperuric-acidemia in eczema and pruritus, and our figures confirm our former observations. We believe that in most instances this increase of the nitrogenous constituents of the blood is due to an improper diet or to some form of renal dysfunction, perhaps a low grade interstitial nephritis. The fact that this occurs more often in subjects who are in middle life or older would seem to favor this view. It is not astonishing that an increase of nitrogenous waste products in the blood should cause an itching of the skin or should at least play a part in the development and perpetuation of eczema.

Nonprotein Nitrogen.—The maximum normal of nonprotein nitrogen in the blood is 40 mg. per hundred cubic centimeters of blood. Twenty-two and six-tenths per cent of the subjects classed as suffering from eczema had 40 mg. or more. This figure equaled that of patients with pruritus, but was less than that for patients with generalized pruritus, for whom it was 36 per cent.

In psoriasis, urticaria, acne, acne rosacea and other diseases it was perceptibly less. The weakness of the figures in diseases other than eczema and pruritus lies in the fact that their number is relatively small.

Urea Nitrogen.—The maximum normal amount of blood urea nitrogen, according to the method employed by us, is 20 mg. per hun-

dred cubic centimeters of blood. A pathologic increase in blood urea nitrogen is evidence of renal dysfunction.

Twenty-one per cent of 452 cases of eczema showed 20 mg. or more of urea nitrogen in the blood. Pruritus exhibited 26 per cent among 141 cases. In thirty-six cases of generalized pruritus, the proportion was 44.4 per cent. In other dermatoses it was perceptibly lower, although here again reference must be made to the relative sparsity of cases.

TABLE 1.—*Percentages of Cases Showing an Increase in Nitrogenous Constituents of the Blood*

A. Nonprotein Nitrogen

Condition	Number of Cases Showing Increase (40 Mg. or More)	Number of Cases Observed	Per Cent Showing Increase	
Psoriasis	6	50	12.0	
Pruritus	31	137	22.6	(In generalized pruritus, 12 of 36, or 36.1%)
Urticaria	4	25	16.0	
Acne	1	7	14.3	
Acne rosacea	2	11	18.2	
Eczema	101	448	22.6	

B. Urea Nitrogen

Condition	Number of Cases Showing Increase (20 Mg. or More)	Number of Cases Observed	Per Cent Showing Increase	
Psoriasis	8	52	15.4	
Pruritus	37	141	26.2	(In generalized pruritus, 16 of 36, or 44.4%)
Urticaria	2	24	8.4	
Acne	1	9	11.1	
Acne rosacea	1	11	9.1	
Eczema	105	452	21.0	

C. Uric Acid

Condition	Number of Cases Showing Increase (4 Mg. or More)	Number of Cases Observed	Per Cent Showing Increase	
Psoriasis	17	52	32.7	
Pruritus	59	143	41.3	(In generalized pruritus, 17 of 37, or 45.9%)
Uritcaria	7	25	28.0	
Acne	2	10	20.0	
Acne rosacea	3	11	27.3	
Eczema	217	455	47.7	

Uric Acid.—There is some difference of opinion as to the maximum normal amount of uric acid in the blood. In order to have a figure which would surely not be too low and which would be regarded as pathologic by most authorities, we selected 4 mg. per hundred cubic centimeters of blood. The method employed was devised by one of us (H. B.) and was thoroughly checked up against other methods.

Of 455 cases of eczema, 217, or 47.7 per cent, showed 4 mg. or more per hundred cubic centimeters of blood; the highest amount found was 7.6 mg. Of 143 cases of pruritus, fifty-nine, or 41.3 per cent, showed an excess; of thirty-seven cases of generalized pruritus, seventeen, or 45.9 per cent, showed an excess; of fifty-two cases of psoriasis, seventeen, or 32.7 per cent, were pathologic. In other dermatoses, the uric acid was perceptibly less.

TABLE 2.—*Chemical Blood Studies in Eczema*

Patient	Condition	Age	Sex	Nonprotein Nitrogen	Urea Nitrogen	Dextrose	Uric Acid	Comment
K. S. G.	Eczema	44	F	34	13	81	4.0	Eyelids and hands
B.	Eczema	55	F	44	20	95	5.1	Palms
E.	Eczema	50	M	45	25	70	4.9	Recurrent on hands
F.	Eczema	59	M	38	15	133	3.5	Positive Wassermann
G. A. B.	Eczema	36	F	38	15	78	3.4	Recurrent
I. F.	Eczema	49	M	46	21	82	4.0	
T.	Eczema	35	M	42	17	79	2.6	Recurrent; fingers
K.	Eczema	30	F	35	15	108	2.1	Hand
H.	Eczema	60	M	43	21	114	6.3	Face; recurrent
P.	Eczema	76	M	40	21	104	3.5	Hands
D. L.	Eczema	56	M	52	26	80	5.2	Chest and abdomen
A. W.	Eczema	42	M	46	20	94	4.6	Recurrent; hands
J.	Eczema	20	F	46	20	96	2.9	Since childhood
D.	Eczema	22	F	40	18	80	2.2	
K.	Eczema	30	F	46	21	85	4.0	Acute
L.	Eczema	41	F	29	12	115	2.2	Generalized
E. S. C.	Eczema	47	M	42	19	93	4.5	Hands
C. S.	Eczema	65	M	33	15	146	6.2	At intervals for 20 years
D. P.	Eczema	16	F	37	12	90	2.1	Since infancy
C.	Eczema	60	M	40	15	93	4.2	Scrotum; chronic
M. B. F.	Eczema	53	F	38	19	110	3.6	Hands
I. P. M.	Eczema	39	M	36	16	96	4.3	Duration, 30 years
T. R.	Eczema	34	F	30	15	102	2.9	Chronic; face
H. B.	Eczema	20	M	35	15	100	3.5	
H. B. G.	Eczema	42	M	35	17	104	4.9	
M. T. B.	Eczema	49	F	34	16	95	3.0	
M. P.	Eczema	70	F	32	11	98	2.8	Ankles
J. N.	Eczema	18	M	36	17	90	4.2	Severe; long standing
D.	Eczema	55	M	..	..	64	2.7	
L. K.	Eczema	49	F	35	15	98	4.3	Neck and face; severe itching
K.	Eczema	32	F	38	18	98	2.6	Mild; fingers
L.	Eczema	51	M	37	19	104	3.8	Acute
O. C.	Eczema	63	F	26	12	102	2.4	
L.	Eczema	32	F	29	14	100	1.0	
S. H. L.	Eczema	28	F	29	15	106	2.8	
C.	Eczema	64	M	34	18	100	3.7	
K.	Eczema	51	M	35	16	83	5.5	Also latent syphilis
J. R.	Eczema	62	M	27	10	100	3.5	Vesicular
H.	Eczema	51	F	28	13	100	2.3	
L. H.	Eczema	51	M	36	17	84	5.9	Scalp
B.	Eczema	73	M	42	22	114	4.5	
F. K.	Eczema	35	M	27	11	80	4.3	
B.	Eczema	42	F	35	..	85	5.3	
J. M.	Eczema	40	F	34	18	95	4.8	Pruritic patch
E. S. S.	Eczema	48 ?	F	27	11	93	3.0	Vesicular; fingers
O. D.	Eczema	40	M	32	16	102	3.3	
S.	Eczema	25	F	30	14	94	2.8	
J. H. D.	Eczema	60	M	40	19	106	3.9	Chronic; blood pressure, 195
B. H.	Eczema	11	F	32	14	98	2.4	
C. K.	Eczema	52	M	36	15	118	3.4	Hands
J. R.	Eczema	57	M	39	19	121	2.5	
S. B.	Eczema	45	M	30	14	96	3.1	
B.	Eczema	70	F	38	20	273	3.2	Also pruritus vulvae
J. H.	Eczema	34	M	38	17	100	3.9	Face
P. R.	Eczema	78	M	28	13	104	4.3	Ankles and hands
E. L.	Eczema	22	F	35	14	90	2.4	
S.	Eczema	45	F	35	16	112	2.3	Hands
W. B.	Eczema	67	M	35	15	110	4.5	
I. H.	Eczema	33	M	32	13	95	3.6	Severe; recurrent
A. S.	Eczema	43	M	30	14	106	4.9	
E. B.	Eczema	59	M	36	16	110	2.4	
J. S.	Eczema	43	M	32	13	106	3.4	
F. C. T.	Eczema	48	F	32	13	100	2.4	
L. S. K.	Eczema	49	M	33	12	125	3.1	
E. L. S.	Eczema	54	M	35	14	110	3.8	
R. E.	Eczema	18	M	29	11	90	3.1	
J. H.	Eczema	60	M	37	18	94	3.8	
H. C. D.	Eczema	41	M	33	14	90	4.8	
J. B.	Eczema	42	M	34	15	98	3.6	
E. C.	Eczema	42	F	30	13	104	2.6	
D. A. D.	Eczema	47	M	43	20	90	4.1	
P. B.	Eczema	50	M	46	28	92	5.3	Erythematopapular on trunk
B. F.	Eczema	79	F	39	20	103	2.2	Hands
W. K.	Eczema	42	M	60	16	86	6.4	On the trunk
W. S.	Eczema	17	M	35	18	95	4.6	Chronic
E. C.	Eczema	66	F	34	15	128	5.8	
F. G.	Eczema	58	F	30	14	96	4.7	
J. M. S.	Eczema	62	F	32	15	130	5.6	Erythematosquamous
A. I. R.	Eczema	27	M	30	13	108	2.8	

TABLE 2.—*Chemical Blood Studies in Eczema—Continued*

Patient	Condition	Age	Sex	Nonprotein Nitrogen	Urea Nitrogen	Dextrose	Uric Acid	Comment
L.	Eczema	52	M	34	15	102	2.7	
H. W.	Eczema	69	M	39	20	96	4.4	
D. S.	Eczema	35	M	30	14	103	1.8	
W. F.	Eczema	73	M	30	15	100	3.3	
D. H.	Eczema	54	M	30	14	112	4.8	
L. K.	Eczema	30	M	36	18	100	2.7	
I. B.	Eczema	39	M	40	19	88	3.3	
L.	Eczema	42	F	30	13	98	2.2	Palms and soles
A. Mc.	Eczema	40	M	32	13	101	5.5	Rebellious and severe
M. S.	Eczema	40	M	41	21	102	3.3	
E. L. B.	Eczema	44	F	30	14	110	3.9	
H. T. D.	Eczema	51	M	38	16	106	3.3	
T. D.	Eczema	10	F	32	13	106	2.0	
T.	Eczema	40	M	40	19	118	3.3	
G. M. A.	Eczema	26	F	28	12	110	2.0	
R.	Eczema	40	F	34	15	110	3.6	
H. S.	Eczema	47	F	38	19	101	2.3	
S. H. H.	Eczema	45	M	38	18	100	5.0	Chronic; hands
J. S. M.	Eczema	42	F	32	14	102	2.4	
S. R.	Eczema	32	F	32	15	96	2.7	
C. L.	Eczema	45	F	41	22	104	3.3	
J. S.	Eczema	42	F	..	18	...	3.0	
M. B. D.	Eczema	38	M	39	20	100	3.9	
B. C.	Eczema	47	F	36	18	116	3.3	
M. S.	Eczema	30	F	41	22	110	3.3	
E. J. L.	Eczema	67	M	53	23	47	2.9	
T. B. G.	Eczema	70	M	35	..	104	5.0	
H. T. H.	Eczema	22	M	36	18	100	3.0	
H. B.	Eczema	38	M	35	17	102	4.0	Also pruritis
S. H.	Eczema	18	F	35	17	97	2.8	Since childhood
J. F. K.	Eczema	63	M	38	19	104	4.6	
M. B.	Eczema	30	M	34	16	100	3.8	
E. S.	Eczema	14	F	36	16	...	3.7	
M. H. S.	Eczema	32	M	40	21	...	4.0	
A. K.	Eczema	40	F	30	11	110	3.2	
S.	Eczema	23	F	28	11	...	2.1	Fissured fingers
C. W.	Eczema	22	M	38	19	108	3.8	
H. S.	Eczema	52	F	38	19	96	3.1	
S. L. L.	Eczema	67	M	37	18	90	4.5	
H. M.	Eczema	31	M	44	23	100	5.5	Vesicular; hands and feet
W. R.	Eczema	28	M	35	17	102	3.6	
R. J. C.	Eczema	59	F	32	15	115	4.6	
M. S.	Eczema	53	M	34	16	100	3.7	
D. H. F.	Eczema	36	M	35	16	110	4.5	
M.	Eczema	21	F	..	14	...	2.0	
J. R.	Eczema	29	M	30	14	100	4.1	Severe; recurrent
F. B. G.	Eczema	21	M	31	13	92	2.9	
J. G. H.	Eczema	40	M	34	16	96	4.1	
B. F.	Eczema	38	M	32	16	67	3.5	
A. A.	Eczema	65	M	34	16	97	2.3	Acute; face
H. W. B.	Eczema	32	M	32	16	88	3.0	
D. B.	Eczema	45	F	30	12	93	2.5	
A. F. B.	Eczema	47	F	22	11	114	1.8	Acute
H. S.	Eczema	55	M	34	16	110	3.3	
L. R. D.	Eczema	38	M	38	18	102	2.9	
S. T.	Eczema	47	F	26	12	100	2.0	
E. E. R.	Eczema	37	F	30	13	101	1.8	
J. H. B.	Eczema	51	M	32	15	83	1.8	Erythematous
H.	Eczema	65	F	34	11	115	1.7	Squamous; hands
V. L.	Eczema	51	M	34	14	114	2.1	
O. K.	Eczema	42	M	30	14	110	2.7	
H. S.	Eczema	24	M	30	11	91	2.3	
C. G.	Eczema	40	M	29	14	91	4.8	
J. L. H.	Eczema	62	M	..	16	...	2.7	Severe
L. S. G.	Eczema	35	M	28	13	90	4.1	
Dr. W.	Eczema	78	M	40	19	210	6.0	
G. W. C.	Eczema	66	M	30	10	80	5.4	
M. C. L.	Eczema	54	F	37	19	92	4.0	Hands and feet
J. C. P.	Eczema	36	M	34	19	90	4.2	Hands
P. P.	Eczema	49	F	30	16	106	3.6	Mild
S. S.	Eczema	52	F	26	12	70	4.4	Vesicular; recurrent
J. J. Mc.	Eczema	66	F	35	15	96	1.5	
C. T.	Eczema	39	F	23	10	92	2.0	
A.	Eczema	50	M	31	17	88	3.2	Hands and feet
J. H.	Eczema	62	F	30	16	81	1.9	
B.	Eczema	42	M	33	14	96	2.0	Severe
D. H.	Eczema	29	F	32	14	97	2.8	
E. B.	Eczema	35	M	32	15	92	4.2	Face, arms and legs

TABLE 2.—*Chemical Blood Studies in Eczema—Continued*

Patient	Condition	Age	Sex	Nonprotein Nitrogen	Urea Nitrogen	Dextrose	Uric Acid	Comment
S. A. M.	Eczema	59	F	28	14	102	2.0	
E. A.	Eczema	62	M	28	11	218	5.4	
G. S.	Eczema	34	M	28	13	106	4.0	
E. T. C.	Eczema	62	M	26	12	88	3.0	
L. H.	Eczema	48	M	28	13	125	4.8	
S. L. G.	Eczema	25	M	42	25	60	5.0	
C. B.	Eczema	40	F	34	13	82	3.3	
W. H. L.	Eczema	38	M	36	17	86	4.6	
J. J. B.	Eczema	70	M	30	15	86	3.0	
N. T.	Eczema	77	M	37	20	71	4.4	
J. G.	Eczema	69	M	37	18	62	4.8	Extensive
E. P.	Eczema	78	F	..	14	...	2.6	
J. O. P.	Eczema	23	F	30	14	76	2.2	
A. M.	Eczema	..	M	39	17	96	3.0	
E. W.	Eczema	62	F	28	13	71	3.2	
E. J. T.	Eczema	44	M	32	15	98	2.5	
T. F. B.	Eczema	53	M	34	18	92	3.7	
E. T.	Eczema	52	F	31	15	97	2.5	
P. V.	Eczema	59	M	..	..	...	5.4	In plaques
J. R.	Eczema	35	F	35	15	108	5.0	Hands
A. C.	Eczema	52	F	35	16	112	3.2	
W. S.	Eczema	20	M	32	16	70	4.0	Recurrent
J. D.	Eczema	22	M	38	18	98	6.4	Chronic
J.	Eczema	35	F	34	17	96	3.4	
W. T. B. R.	Eczema	73	M	35	19	354	4.5	Recurrent
H.	Eczema	43	F	32	15	80	4.2	
R. G.	Eczema	23	M	32	14	82	2.6	
E. P. P.	Eczema	55	M	39	21	102	5.0	
W.	Eczema	60	M	34	17	105	3.6	
A. D. C.	Eczema	56	F	34	17	87	2.0	
C. B.	Eczema	40	F	32	16	64	2.0	
I. L.	Eczema	50	M	48	28	139	3.0	
E. B. M.	Eczema	61	M	32	15	76	2.7	
H. T. W.	Eczema	37	F	33	17	104	1.8	
C. C.	Eczema	71	F	36	18	88	4.8	
W. R.	Eczema	65	M	39	19	81	4.5	
S. N.	Eczema	47	M	41	19	95	3.3	Severe; chronic
M. F.	Eczema	20	F	33	15	100	4.0	
E. D.	Eczema	58	F	42	20	118	4.1	
B. F.	Eczema	42	F	39	20	88	3.6	Hands, recurrent
H. R.	Eczema	52	F	44	23	102	3.2	
R. R. K.	Eczema	32	M	35	18	100	3.0	
C. K.	Eczema	31	M	31	17	98	3.3	
C. A. C.	Eczema	33	M	30	17	111	4.1	
J. C.	Eczema	65	F	45	23	95	6.2	
B. W.	Eczema	30	M	36	16	118	5.0	
J. F. B.	Eczema	48	M	36	18	67	3.6	
C. C. M.	Eczema	63	F	36	19	87	3.9	
S. S.	Eczema	23	M	33	13	95	4.7	
A. O.	Eczema	20	F	31	15	75	3.4	
Dr. N. E. L.	Eczema	46	M	33	15	100	5.1	
J. K.	Eczema	30	F	31	14	87	3.6	
L. L.	Eczema	15	F	42	22	89	4.4	
E. L. P.	Eczema	15	F	30	11	86	3.7	
P. R. N.	Eczema	41	M	33	17	95	4.1	
R. T. M.	Eczema	47	M	38	22	100	4.0	
H. A. J.	Eczema	60	M	30	14	80	4.0	
J. L. L.	Eczema	46	M	31	14	91	4.1	
F. E.	Eczema	34	F	30	22	105	3.9	
M. G.	Eczema	15	F	36	19	105	2.4	Chronic; allergic type
J. S.	Eczema	68	F	34	14	97	4.3	Refractory eczema of the auditory canals
C. R.	Eczema	32	F	32	18	92	3.5	Chronic
F. L.	Eczema	44	F	35	19	102	4.0	
H. C.	Eczema	..	F	31	16	102	4.6	
B. K.	Eczema	48	F	32	17	100	2.4	
M. C.	Eczema	63	M	38	20	100	3.4	Hand
T. W. S.	Eczema	50	M	28	14	92	4.9	
D. L.	Eczema	72	M	50	33	92	4.0	
A. E.	Eczema	40	F	42	22	108	3.0	
J. C.	Eczema	69	M	47	25	91	3.5	
M. S. W.	Eczema	47	F	34	14	45 ?	3.3	
O. B. S.	Eczema	27	F	34	17	89	4.0	
M. L.	Eczema	46	F	35	18	93	3.8	
Dr. Z.	Eczema	60	M	40	21	125	3.3	
C. W.	Eczema	34	F	35	18	108	2.6	
M. M.	Eczema	58	F	37	19	100	2.6	
M. F.	Eczema	39	M	36	17	66	3.7	
J. J. L.	Eczema	31	M	40	22	98	4.0	
G. C. K.	Eczema	62	M	38	14	106	4.6	

TABLE 2.—*Chemical Blood Studies in Eczema—Continued*

Patient	Condition	Age	Sex	Nonprotein Nitrogen	Urea Nitrogen	Dextrose	Uric Acid	Comment
W. H. G.	Eczema	36	F	36	17	98	3.3	
L. H. M.	Eczema	58	M	41	24	88	4.8	
N. P. C.	Eczema	55	M	39	18	95	4.0	
B. B.	Eczema	52	M	28	14	100	4.5	
A. B.	Eczema	58	F	..	12	125	...	
O. S.	Eczema	52	F	28	14	111	4.8	
A. M.	Eczema	60	F	39	18	100	4.4	
L. M. B.	Eczema	53	M	39	22	133	4.1	Vesicular
L. T.	Eczema	25	M	32	16	95	4.8	
C. H. G.	Eczema	74	M	39	22	132	3.4	
S. D. L.	Eczema	58	F	..	16	117	2.7	
Dr. G. B. P.	Eczema	57	M	36	18	100	3.6	
H. C. B.	Eczema	30	M	40	22	111	3.5	
J. S. K.	Eczema	69	F	56	30	133	5.0	
M. F.	Eczema	20	F	41	21	111	3.5	Chronic
H. A. J.	Eczema	60	M	45	22	105	4.8	
S. S.	Eczema	17	F	34	15	100	3.5	
J. F. W.	Eczema	60	M	39	22	125	5.2	
M. C.	Eczema	63	M	42	22	133	3.6	
G. K.	Eczema	45	F	31	17	91	2.8	
T. U.	Eczema	87	M	44	21	111	3.5	
F. A. P.	Eczema	47	M	43	23	107	3.2	
J. S.	Eczema	21	M	38	17	100	3.4	
G. S.	Eczema	52	M	36	19	100	2.5	
H. A.	Eczema	63	M	30	22	125	5.7	
W. T. B.	Eczema	38	F	34	17	125	1.5	
R. D.	Eczema	16	F	30	12	105	3.6	
E. S.	Eczema	82	M	32	14	174	3.6	Diabetes
G. H.	Eczema	60	M	39	20	95	6.3	Palm (;)
P. R.	Eczema	..	M	44	20	102	4.9	
G. W. M.	Eczema	53	F	30	44	90	6.2	Later, 5.5
A. A. B.	Eczema	19	F	36	15	95	1.9	
J. S.	Eczema	77	M	31	14	96	3.6	
K. G.	Eczema	58	M	34	13	90	4.0	
R. Mc.	Eczema	36	F	28	13	92	2.5	
G. G.	Eczema	50	M	45	21	118	5.2	
J. H.	Eczema	31	M	33	17	100	2.7	
L. W.	Eczema	46	M	31	11	95	4.0	
F. S.	Eczema	50	F	37	17	91	5.0	
G. D. N.	Eczema	58	M	28	15	77	4.7	
L. M. W.	Eczema	34	F	36	17	98	2.7	
M. S.	Eczema	38	M	35	22	96	4.8	
B.	Eczema	17	M	34	19	101	4.0	
K. O.	Eczema	57	F	34	16	98	3.6	
A. B.	Eczema	56	M	42	22	101	4.6	
W. D.	Eczema	32	M	31	16	102	5.8	
J. W.	Eczema	34	M	30	12	101	2.9	
H. M. N.	Eczema	38	M	33	15	95	5.3	
A. B.	Eczema	60	M	32	17	88	3.0	
M. W.	Eczema	43	M	42	23	98	3.5	
V. C.	Eczema	60	M	31	17	94	4.6	
J. E. W.	Eczema	..	M	39	17	103	4.8	
K. A.	Eczema	20 ?	M	28	11	88	4.0	
W. F. S.	Eczema	..	F	38	20	97	4.7	
L. H. M.	Eczema	58	M	42	20	97	3.7	
M. B.	Eczema	52	F	45	20	100	3.1	
W. T. B.	Eczema	65	M	51	26	400	4.6	
G. H.	Eczema	64	M	44	23	100	5.4	
E. W. C.	Eczema	65	M	37	22	70	6.3	
E. A. D.	Eczema	71	F	..	..	303	...	
W. H. S.	Eczema	26	M	30	13	71	2.6	
F. K.	Eczema	33	F	52	25	...	5.4	
B. F.	Eczema	50	F	30	16	100	3.6	
J. J. E.	Eczema	56	M	40	19	85	4.4	
A. Mc.	Eczema	40	M	38	19	40 ?	5.9	Chronic; recurrent
H. Z.	Eczema	18	M	30	14	95	4.7	
W. B.	Eczema	75	M	44	23	230	5.7	Diabetes
E. W. C.	Eczema	65	M	35	18	62	6.1	
J. M.	Eczema	38	M	29	15	85	4.0	
Dr. C. M.	Eczema	58	M	38	19	80	7.1	
Dr. L.	Eczema	54	M	29	12	95	4.8	
H. V.	Eczema	16	M	33	12	70	4.0	
L. K.	Eczema	30	M	42	24	97	2.5	
F. K.	Eczema	20	M	38	17	52 ?	4.6	
L. D.	Eczema	56	M	33	17	80	2.7	Trade dermatitis.
A. Z.	Eczema	40	F	36	18	84	3.0	
A. M. S.	Eczema	..	F	35	17	90	6.1	
H. M.	Eczema	58	F	40	22	97	4.3	Hands
H. A.	Eczema	28	F	36	14	100	2.9	

TABLE 2.—*Chemical Blood Studies in Eczema—Continued*

Patient	Condition	Age	Sex	Nonprotein Nitrogen	Urea Nitrogen	Dextrose	Uric Acid	Comment
C.	Eczema	52	F	33	14	125	5.3	
L. S.	Eczema	10	F	26	12	121	4.0	
M. B.	Eczema	58	M	..	..	98	5.4	
M. E. W.	Eczema	30	F	40	28	118	3.8	
A. S.	Eczema	40	M	33	17	95	4.8	
R. M. W.	Eczema	47	M	31	16	100	4.2	
H. D. Mc.	Eczema	75	F	42	19	125	5.5	
B. T. Z.	Eczema	37	M	35	18	85	3.1	
H. E. B.	Eczema	..	M	40	22	95	6.7	
H. M.	Eczema	75	F	38	18	111	5.6	
A. F.	Eczema	55	M	50	27	113	5.0	
A. F.	Eczema	55	M	42	22	97	4.6	
A. F.	Eczema	55	M	45	24	109	5.0	
A. F.	Eczema	55	M	48	28	125	4.5	
M. A.	Eczema	49	F	34	15	108	3.7	
W. D.	Eczema	12	M	42	19	100	4.1	
S. D.	Eczema	42	F	45	22	100	4.5	
Dr. E. E. C.	Eczema	40	M	43	25	167	5.0	Later 4.7, sugar 134
G. W.	Eczema	36	M	39	16	109	5.4	Severe and inveterate; universal
D. F.	Eczema	21	F	30	14	97	2.4	
Dr. M. D.	Eczema	35	M	35	18	190	4.0	
W. M. S.	Eczema	60	F	33	15	100	3.5	
T. G. M.	Eczema	50	F	35	17	111	4.5	
A. F.	Eczema	40	F	50	23	105	4.1	
H. R.	Eczema	70	M	60	36	109	7.6	
F. P.	Eczema	11	F	36	19	118	4.5	
A. D.	Eczema	52	F	50	28	125	7.1	Later, 6.3
C. M.	Eczema	19	M	40	18	100	4.2	
F. O.	Eczema	32	F	32	14	95	3.5	
G. H.	Eczema	23	F	53	25	...	4.9	
E. D.	Eczema	35	M	36	17	80	3.8	
T. K.	Eczema	65	M	56	31	97	5.0	
C. G.	Eczema	67	M	29	13	88	3.6	
M. K.	Eczema	20	M	59	22	91	4.2	On forehead
A. P.	Eczema	24	M	54	26	82	5.1	
P. M.	Eczema	19	M	25	11	96	2.8	
H.	Eczema	40	M	38	18	99	4.0	
B. G.	Eczema	22	F	30	14	100	3.3	Also had pruritis
C. W.	Eczema	67	M	34	18	112	5.5	
B.	Eczema	31	M	36	16	100	3.0	
J. W. A.	Eczema	61	F	28	14	108	2.5	
L. T. A.	Eczema	63	M	32	16	96	5.8	
W. B.	Eczema	28	M	30	14	100	2.8	Legs; duration, 20 years
W. K.	Eczema	71	M	36	18	106	4.6	
T. P.	Eczema	65	M	..	..	...	5.5	
(?)	Eczema	31	F	..	..	...	2.6	Moist
S. B.	Eczema	78	F	39	20	82	4.0	
I. L.	Eczema	50	M	43	22	111	2.8	
A. B.	Eczema	41	M	39	17	99	5.7	
S. D.	Eczema	60	F	25	12	107	4.1	
S. K. M.	Eczema	61	F	36	15	96	2.6	
A. B.	Eczema	39	M	25	13	97	4.9	
W. A.	Eczema	56	M	44	20	97	5.5	Acute
F. L.	Eczema	39	F	39	18	99	2.8	Severe
A. B.	Eczema	72	F	35	12	100	5.0	
A. E. G.	Eczema	50	M	..	..	102	2.4	
M.	Eczema	55	F	33	14	100	3.0	
R. S.	Eczema	73	M	45	22	121	4.6	
R. G. B.	Eczema	38	M	45	23	102	5.0	
W. H.	Eczema	..	F	35	17	103	4.0	
A. J. S.	Eczema	..	M	27	12	97	3.4	
H. R.	Eczema	64	M	29	13	100	3.7	
D. J. Mc.	Eczema	51	M	39	14	111	4.0	
M. K.	Eczema	50	F	31	14	87	3.6	
M. L.	Eczema	72	M	40	22	105	3.0	
S. R.	Eczema	52	F	41	23	118	5.0	
W. F.	Eczema	49	M	28	14	148	5.3	
O. M.	Eczema	66	F	34	16	91	4.3	
D. T. M.	Eczema	77	M	40	22	90	4.8	
S. L. H.	Eczema	67	M	36	17	104	4.3	
B. T. O.	Eczema	57	F	40	20	105	4.9	
E. C. R.	Eczema	24	M	28	12	102	1.5	
Y. G.	Eczema	28	F	26	12	97	2.7	
M. B.	Eczema	23	F	30	15	76	2.4	
P.	Eczema	57	M	40	21	91	4.8	
D. P.	Eczema	41	F	30	14	80	2.5	
T. D. I.	Eczema	75	M	34	17	126	3.5	
C. A.	Eczema	38	M	25	10	96	2.0	
J. S.	Eczema	58	M	30	14	77	5.4	
W.	Eczema	50	M	28	14	80	5.0	

TABLE 2.—*Chemical Blood Studies in Eczema—Continued*

Patient	Condition	Age	Sex	Nonprotein Nitrogen	Urea Nitrogen	Dextrose	Uric Acid	Comment
B. A.	Eczema	12	M	20	13	92	3.2	Chronic
F. C. G.	Eczema	24	M	34	17	90	5.3	On feet
D.	Eczema	22	M	..	..	...	4.6	
G.	Eczema	76	M	28	14	89	3.0	
W. J. K.	Eczema	74	M	30	15	122	3.2	
J. R. X.	Eczema	61	M	35	17	85	5.7	Chronic; universal
M. F.	Eczema	52	M	..	..	89	3.4	
F.	Eczema	52	M	35	18	74	2.3	Vesicular
J. O.	Eczema	..	F	36	17	89	2.2	
E. M.	Eczema	61	F	28	13	94	2.4	
L. F.	Eczema	27	F	23	11	96	2.4	
I. R.	Eczema	46	F	36	18	89	2.7	
J. M.	Eczema	80	M	42	24	100	3.6	
C. Y. N.	Eczema	31	M	22	9	62	2.4	
S.	Eczema	50	F	20	8	89	3.1	
S.	Eczema	19	M	28	10	75	3.1	Later 3.6 and 4.3
S. S.	Eczema	63	M	45	26	92	3.1	Hands
B. R. B.	Eczema	67	F	30	13	122	3.1	Later, uric acid 4.0 and 3.9
D. F. B.	Eczema	63	M	28	10	103	4.1	Erythematosis
M. B.	Eczema	51	F	36	16	97	5.3	
P.	Eczema	48	F	31	14	122	3.0	Acute
A. D.	Eczema	38	M	43	17	99	3.2	Recurrent
R.	Eczema	18	F	34	19	107	3.7	Chronic
R. W.	Eczema	59	M	47	21	80	6.3	Chronic; recurrent
I. L.	Eczema	43	M	31	16	18	4.1	Chronic; severe
H.	Eczema	47	M	40	20	132	5.9	Chronic; severe; recurrent
H. S. R.	Eczema	62	M	37	18	123	4.5	Recurrent on hands
McH.	Eczema	68	M	46	15	90	4.8	Severe; acute
M.	Eczema	52	F	41	20	88	2.9	Hands and feet
J. S. S.	Eczema	56	M	42	15	83	3.4	Mild
J. S. C.	Eczema	55	M	..	..	...	4.8	
O. F. C.	Eczema	54	F	31	16	105	3.3	
J. A. H.	Eczema	54	M	36	14	97	4.2	
C. H. S.	Eczema	40	M	34	17	100	2.6	
L. W. C.	Eczema	46	M	33	14	95	5.0	
T. T.	Eczema	62	M	38	17	100	2.5	
A. G. S.	Eczema	53	M	32	14	100	4.0	
Dr. T. W.	Eczema	57	M	35	20	98	5.0	
W. F.	Eczema	58	M	31	14	94	4.6	
H. A. L.	Eczema	60	F	40	21	91	5.0	
M. D.	Eczema	54	F	28	12	95	3.5	
H. L.	Eczema	67	M	40	17	111	2.6	
S. W.	Eczema	58	F	37	17	100	3.0	
Dr. S. C.*	Eczema	30	M	28	11	95	4.2	
J. F.	Eczema	60	M	41	25	125	5.0	
A. T. S.	Eczema	51	F	42	20	97	4.0	
P. S. S.	Eczema	54	M	36	20	95	5.0	
A. S.	Eczema	40	M	..	..	...	4.7	
H. E.	Eczema	25	M	30	13	100	2.9	
R. J. H.	Eczema	34	M	35	22	100	3.6	
B. G.	Eczema	50	M	37	17	98	5.5	
J. R. H.	Eczema	51	M	30	14	80	5.0	
F. L. F.	Eczema	57	M	39	21	106	2.4	
C. G. B.	Eczema	45	F	42	20	100	5.1	
C. C. A.	Eczema	40	M	36	14	95	5.6	Hands and buttocks
M. G.	Eczema	36	F	28	..	95	3.0	
H. K.	Eczema	49	M	..	..	100	3.1	
M. W.	Eczema	48	F	12	26	95	4.5	
C. R. C.	Eczema	57	F	35	17	95	1.7	
R. B. H.	Eczema	36	M	34	14	80	3.1	
D. A.	Eczema	33	M	34	17	100	5.0	
R. L. M.	Eczema	46	M	35	17	94	3.0	
D. B.	Eczema	30	M	..	..	...	4.5	
M. F. L.	Eczema	46	F	37	19	100	3.0	
M. E. F.	Eczema	58	M	31	14	103	4.5	
S. H.	Eczema	40	M	33	16	96	3.8	
T. G.	Eczema	59	M	..	17	...	6.2	
G. W. D.	Psoriasis	39	M	34	17	70	3.4	
C. M.	Psoriasis	44	M	32	15	102	4.3	
W.	Psoriasis	25	M	23	15	74	4.1	
A. H. W.	Psoriasis	36	M	32	12	116	5.0	
R. B.	Psoriasis	32	M	32	15	89	3.7	
J. B.	Psoriasis	18	F	32	15	81	4.0	
M.	Psoriasis	48	M	29	14	108	2.8	
J. W. M. K.	Psoriasis	53	M	36	18	104	3.4	
L. A. M.	Psoriasis	59	F	34	17	77	3.0	
J. C. S.	Psoriasis	55	M	37	15	100	3.7	
F. R.	Psoriasis	..	F	28	11	100	2.4	
W. A. C.	Psoriasis	61	M	42	21	67	5.3	

TABLE 2.—*Chemical Blood Studies in Psoriasis and Generalized Pruritus*

Patient	Condition	Age	Sex	Nonprotein Nitrogen	Urea Nitrogen	Dextrose	Uric Acid	Comment
J. A. S.	Psoriasis	48	M	36	19	148	3.4	
A. E. K.	Psoriasis	42	F	34	17	571	2.6	Diabetes
E. M.	Psoriasis	75	F	28	14	111	5.0	Diabetes
A. F.	Psoriasis	56	F	32	15	95	4.3	Arthritis
L. P.	Psoriasis	40	M	36	17	105	5.4	
M. W.	Psoriasis	65	F	40	21	82	3.0	
L. F.	Psoriasis	40	M	32	15	146	5.6	Universal; also arthritis; later, sugar 115
A. P.	Psoriasis	45	M	46	26	105	6.1	
F. T. P.	Psoriasis	50	F	42	20	80	5.2	
A. A.	Psoriasis	..	M	42	22	131	5.0	
W. B. S.	Psoriasis	25	M	33	14	87	3.0	
L. C.	Psoriasis	26	F	..	17	87	2.9	
F. J. E.	Psoriasis	54	F	33	17	270	4.1	
E. C.	Psoriasis	66	F	39	20	131	3.2	
L. D.	Psoriasis	60	M	27	13	113	2.7	
A. F.	Psoriasis	35	M	25	11	182	3.2	
L. J.	Psoriasis	18	M	26	9	65	2.5	
M. G.	Psoriasis	44	F	27	14	118	3.3	
J. A. H.	Psoriasis	29	M	29	11	111	2.8	
H.	Psoriasis	60	F	33	16	90	4.5	
R. S. I.	Psoriasis	38	F	38	18	98	2.5	
E. H.	Psoriasis	63	M	46	23	88	5.2	
W.	Psoriasis	45	F	39	14	100	3.2	
N. D.	Psoriasis	57	F	38	18	100	3.8	
C. M. C.	Psoriasis	57	M	31	13	160	3.1	Later, dextrose was 147
P. R.	Psoriasis	35	F	32	11	102	2.2	
E. S.	Psoriasis	56	M	39	19	107	4.2	
O. C.	Psoriasis	38	F	30	13	114	3.1	
M. P. W.	Psoriasis	35	M	38	19	97	4.8	
C.	Psoriasis	40	M	30	14	96	2.3	
F. S.	Psoriasis	52	M	..	13	...	3.5	
M. M.	Psoriasis	23	M	30	15	107	3.5	
J. H.	Psoriasis	48	M	36	15	107	2.4	
Dr. B.	Psoriasis	29	M	39	23	109	3.7	
L. W. F.	Psoriasis	22	M	25	10	108	2.8	
K. W.	Psoriasis	32	F	28	16	95	2.5	
F. R. S.	Psoriasis	34	M	27	11	95	2.4	
M. P.	Psoriasis	31	M	32	14	80	2.0	
W. B.	Psoriasis	46	F	28	15	96	3.5	
P. W.	Psoriasis	27	M	27	12	90	2.5	
M. V.	Generalized pruritus	35	M	30	20	100	2.8	
H.	Generalized pruritus	62	F	37	17	102	1.7	
W. A. C.	Generalized pruritus	75	F	39	22	101	6.1	
J. B.	Generalized pruritus	77	M	41	24	67	5.2	
R. B.	Generalized pruritus	25	M	28	14	99	2.0	
R. L.	Generalized pruritus	62	F	33	..	96	2.6	
T.	Generalized pruritus	53	F	28	14	92	2.7	Cholesterol, 225
I. W.	Generalized pruritus	65	M	40	23	100	3.4	
Dr. S. L.	Generalized pruritus	59	M	34	14	93	4.8	
B. E. L.	Generalized pruritus	37	M	40	22	88	5.0	
M. V.	Generalized pruritus	33	F	22	14	60 ?	3.1	
T. W.	Generalized pruritus	60	M	33	17	117	4.0	
N. L.	Generalized pruritus	30	M	31	14	44 ?	4.2	
G. A.	Generalized pruritus	..	F	..	..	92	3.0	
S. W.	Generalized pruritus	..	M	35	18	90	2.6	
A. M. W.	Generalized pruritus	..	M	43	28	100	3.9	
H. Z.	Generalized pruritus	70	F	41	21	90	3.2	
M. P.	Generalized pruritus	65	F	31	18	160	4.6	
M. T.	Generalized pruritus	45	F	45	28	131	5.0	
W.	Generalized pruritus	73	M	42	20	114	4.4	
A. S.	Generalized pruritus	47	F	40	22	108	3.3	
W. H. W.	Generalized pruritus	68	M	32	15	113	2.1	
M. P. K.	Generalized pruritus	42	F	37	15	82	4.0	
F. S. L.	Generalized pruritus	52	M	40	19	108	4.0	
J. S.	Generalized pruritus	33	M	32	16	101	4.7	
P. F. H.	Generalized pruritus	60	M	49	26	117	3.5	
S. B.	Generalized pruritus	58	F	40	22	109	4.3	
A. B. G.	Generalized pruritus	69	M	32	15	95	5.0	Also eczema
J. H.	Generalized pruritus	73	M	39	21	80	3.2	
I.	Generalized pruritus	30	M	28	14	55	3.3	
L.	Generalized pruritus	..	F	45	23	71	4.0	
O. R.	Generalized pruritus	31	M	28	13	95	2.5	
L. G.	Generalized pruritus	73	M	54	25	...	...	
T. T.	Generalized pruritus	39	F	36	16	100	4.6	
D. L.	Pruritus hiemalis	78	M	38	18	99	4.7	
G. H. B.	Pruritus hiemalis	53	F	38	19	98	2.4	
C. T. R.	Pruritus hiemalis	53	M	37	19	116	5.1	
L. W.	Pruritus hiemalis	60	M	40	22	100	3.0	

TABLE 2.—*Chemical Blood Studies in Pruritus*

Patient	Condition	Age	Sex	Nonprotein Nitrogen	Urea Nitrogen	Dextrose	Uric Acid	Comment
W. M.	Pruritus hiemalis	..	F	35	17	103	4.0	
O.	Pruritus hiemalis	..	M	26	10	90	2.8	
M. K. N.	Pruritus hiemalis	..	F	32	17	96	3.0	
J. T. Mc.	Pruritus hiemalis	56	M	36	15	96	4.8	
T. H. S.	Pruritus hiemalis	53	M	31	17	130	4.0	
D. L. H.	Pruritus hiemalis	51	M	37	17	90	4.7	
P.	Pruritus	45	M	42	22	40 ?	3.8	
S. P.	Pruritus	21	F	39	19	93	3.3	
E. S.	Pruritus	65	F	41	21	93	3.2	
E. G.	Pruritus	68	M	35	17	97	5.3	
H. K.	Pruritus	72	M	31	15	147	4.0	
E. B.	Pruritus	57	M	40	22	100	5.0	
S. H. B.	Pruritus	61	M	36	18	100	4.5	
	Pruritus	57	M	28	11	100	5.4	
W. M.	Pruritus	85	F	24	14	110	1.0	
K. B.	Pruritus	22	M	30	13	108	5.4	Trunk; severe
Mc.	Pruritus	60	M	37	16	115	3.3	On arms
H.	Pruritus	56	M	40	22	77	3.6	
S. W.	Pruritus	26	F	30	14	80	4.2	Legs and thighs
F. H.	Pruritus	43	F	25	11	102	2.5	
H. M.	Pruritus	48	F	38	19	95	4.1	
B. C.	Pruritus	53	F	30	14	108	2.8	
A. Mc.	Pruritus	65	F	33	17	100	2.6	
J. R.	Pruritus	66	M	56	25	118	3.5	
W. S. T.	Pruritus	54	M	37	18	117	4.7	
W. H. S.	Pruritus	84	M	39	19	100	5.0	
H. W. P.	Pruritus	40	F	38	18	85	3.8	Scalp
J. S.	Pruritus	37	M	29	13	92	3.7	
L. V. Z.	Pruritus	29	F	31	15	100	4.0	
W. H. T.	Pruritus	72	F	32	14	156	4.0	
H. L.	Pruritus	50	M	38	18	77	3.1	
I. S.	Pruritus	34	F	36	17	83	2.5	
H. M. W.	Pruritus	35	M	..	12	82	3.7	Arms and back
R.	Pruritus	32	F	34	15	110	1.5	Arms
F. L. L.	Pruritus	35	F	30	13	80	1.7	Xerosis cutis
T.	Pruritus	29	F	31	15	73	3.1	Scalp, neck and arms
J. S.	Pruritus	58	M	30	14	77	5.4	
G. K.	Pruritus	73	M	39	21	115	2.5	
M. K.	Pruritus	52	M	..	15	91	3.0	
H. C. H.	Pruritus	40	F	38	18	76	3.8	
T. T.	Pruritus	44	M	28	12	104	5.4	Also eczema
K. T. A.	Pruritus	75	M	30	15	92	5.6	Severe
G. C. F.	Pruritus	69	F	30	14	99	4.5	
J. F. S.	Pruritus	56	M	40	22	...	4.8	
M. G.	Pruritus	43	F	41	22	100	5.1	
W. J. W.	Pruritus	46	F	39	20	102	3.2	
F. A.	Pruritus	26	M	34	17	53 ?	3.7	
S. A. M.	Pruritus	36	F	42	23	91	3.4	
S. S.	Pruritus	43	F	38	17	100	...	
R. L. W.	Pruritus	30	M	32	17	111	2.6	Only after bathing
F. P.	Pruritus	48	F	32	16	95	3.2	
R. G.	Pruritus	58	F	32	15	89	2.6	Great excess of bilirubin; developed jaundice
M. D.	Pruritus ani	35	M	35	18	100	5.5	
R.	Pruritus ani	49	F	33	16	90	4.5	
H. G.	Pruritus ani	38	M	42	21	90	6.6	
L.	Pruritus ani	29	M	29	12	102	3.5	
C.	Pruritus ani	34	M	30	13	107	5.0	
C. S. W.	Pruritus ani	27	M	37	18	103	4.1	
S. T.	Pruritus ani	21	M	35	15	96	3.0	
M. S.	Pruritus ani	31	M	26	11	95	2.7	
E. B.	Pruritus ani	40	M	38	14	167	3.2	Also pruritus capitis
Dr. H.	Pruritus ani	56	M	34	16	103	3.7	
F. W. M.	Pruritus ani	40	M	28	14	91	4.3	
F. M.	Pruritus ani	40	M	35	14	100	3.9	
T. P. F.	Pruritus ani	38	M	38	20	50 ?	5.0	
J. F. S.	Pruritus ani	56	M	40	22	...	4.8	
H. W.	Pruritus ani	32	M	42	14	117	3.7	
A. W.	Pruritus ani	51	M	42	22	98	6.8	
T. A. W.	Pruritus ani	43	M	38	18	80	4.2	Also palms and soles
W. E. H.	Pruritus ani	23	M	40	21	100	3.9	
F. M.	Pruritus ani and vulvae	30	F	36	18	95	2.8	
J. B.	Pruritus hiemalis	40	F	42	24	67 ?	3.7	
M. N.	Pruritus hiemalis	47	M	45	23	105	4.8	
J. B.	Pruritus hiemalis	70	M	32	15	98	3.0	
R. L. C.	Pruritus hiemalis	50	M	46	26	92	3.2	
M. D.	Pruritus ictericus	75	F	53	33	125	3.5	Jaundice
R. C.	Pruritus scroti	41	M	32	15	105	4.1	
W. J. G.	Pruritus scroti	20	M	33	17	70	5.2	Also pruritus capitis

TABLE 2.—*Chemical Blood Studies in Pruritus and Other Dermatoses*

Patient	Condition	Age	Sex	Nonprotein Nitrogen	Urea Nitrogen	Dextrose	Uric Acid	Comment
R. C. P.	Pruritus scroti	27	M	32	16	93	3.5	Also anal itching
D. S.	Pruritus scroti	..	M	..	..	...	3.9	
G. L.	Pruritus scroti	59	M	33	14	96	4.7	
Dr. R. A. B.	Pruritus scroti	33	M	30	14	98	4.1	
C.	Pruritus senilis	73	F	27	13	86	3.3	
G. B. Mc.	Pruritus (wrists)	63	M	39	22	90	3.4	
J. K.	Pruritus vulvae	50	F	31	16	111	4.1	
M. K.	Pruritus vulvae	34	F	..	11	103	2.5	
C. D.	Pruritus vulvae	66	F	30	14	90	2.0	
H. K.	Pruritus vulvae	55	F	31	15	308	2.5	Diabetes
S.	Pruritus vulvae	55	F	31	14	111	2.5	
M. G.	Pruritus vulvae	..	F	..	..	100	2.8	
C. W.	Pruritus vulvae	39	F	35	15	100	2.9	
R. G.	Pruritus vulvae	46	F	29	13	94	2.3	
M. L.	Pruritus vulvae	47	F	47	25	88	4.4	
E. M.	Pruritus vulvae	60	F	30	14	108	4.2	
M. E.	Pruritus vulvae	38	F	31	16	295	3.8	
G. H. M.	Pruritus vulvae	36	F	30	19	95	3.6	
M. J.	Pruritus vulvae	37	F	31	13	95	1.8	
P.	Ulcus	..	F	28	14	107	3.8	
A. J.	Urticaria	46	F	38	15	100	3.3	
I. L.	Urticaria	57	M	33	19	91	3.9	Chronic
B. K.	Urticaria	38	M	40	22	70	2.8	
H.	Urticaria	48	F	44	19	97	2.6	
W.	Urticaria	24	M	29	11	110	3.0	
S. A.	Urticaria	39	M	38	19	110	4.7	
O.	Urticaria	54	M	26	11	84	1.4	
F. G. B.	Urticaria	20	M	34	15	80	4.7	Duration, 6 months
H. W.	Urticaria	30	F	22	11	92	5.5	Duration, 4 years
L. T.	Pruritus capitis	26	F	26	11	91	2.3	
P. A.	Pruritus capitis	35	M	29	14	106	2.9	
E. W.	Pruritus capitis	22	F	28	14	85	2.7	
C. K.	Pruritus capitis	45	M	..	..	95	...	
L. E. E.	Pruritus capitis	48	M	34	17	97	3.1	
Mc.	Pruritus corporis	55	M	31	15	100	2.9	
H. S. G.	Pruritus of the face and hands	71	M	34	16	104	3.4	
J. R.	Pruritus of the face and arms	..	M	30	13	109	3.2	
C. H.	Pruritus of the face and body	67	M	36	18	122	5.8	
V. G.	Pruritus of the face	34	M	31	14	98	6.2	Severe; many years' duration
D. W.	Pruritus of the face	19	M	..	16	70	3.4	
R.	Generalized pruritus	31	F	24	10	77	2.5	
F. W. K.	Generalized pruritus	56	F	34	14	86	2.5	
S. G.	Generalized pruritus	49	F	..	9	..	2.4	
R. F.	Generalized pruritus	54	F	39	20	78	5.0	Severe
C. W.	Generalized pruritus	46	M	..	..	105	...	
S. Z.	Urticaria	31	M	29	14	92	4.4	
W. B. J.	Urticaria	31	F	32	15	95	1.8	Chronic
H. S.	Urticaria	27	M	35	16	81	3.2	
M.	Urticaria	50	M	42	22	69	3.9	
W.	Urticaria	43	F	18	7	97	2.4	Chronic
F. H.	Urticaria	48	M	33	13	80	3.4	Angioneurotic edema
V. K.	Urticaria	..	F	37	16	97	4.4	
E. M. J.	Urticaria	45	M	41	..	95	4.7	
H. K. H.	Urticaria	30	M	36	19	114	3.0	
J. H.	Urticaria	53	F	30	11	111	3.8	
J. E. C.	Urticaria	..	M	35	19	98	3.0	
E. W. P.	Urticaria	65	F	37	17	89	3.7	
H. E. K.	Urticaria	57	M	33	17	103	3.2	
W. A. B.	Urticaria	46	M	30	13	108	3.5	
R. P.	Urticaria	25	F	36	17	82	3.6	
E. G.	Urticaria	27	F	28	12	95	4.3	
S. H.	Vitiligo	38	M	34	11	102	3.1	
A. R.	Xanthoma	21	M	27	12	129	1.9	
L.	Xerosis cutis	31	M	..	..	82	3.9	
M. J. L.	Dermatitis seborrhoeica	20	F	34	17	67	2.3	
J. M.	Dermatitis seborrhoeica	40	F	33	15	97	4.1	
M. S.	Dermatitis seborrhoeica	..	..	40	21	100	4.3	
R. B.	Dermatitis seborrhoeica	24	M	35	18	91	4.3	
R. G. J.	Dermatitis seborrhoeica	64	F	37	22	100	4.2	Violent itching
G. W. T.	Dermatitis herpetiformis	30	M	39	20	80	1.7	

TABLE 2.—*Chemical Blood Studies in Miscellaneous Dermatoses*

Patient	Condition	Age	Sex	Nonprotein Nitrogen	Urea Nitrogen	Dextrose	Uric Acid	Comment
T. O.	Dermatitis herpetiformis	39	M	30	14	100	4.3	
J. S.	Dermatitis herpetiformis	40	M	36	18	98	4.3	
R. F.	Dermatitis herpetiformis	33	F	31	11	100	5.4	
K.	Dermatitis (mercurial)	34	M	28	14	76	3.5	
K.	Dermatitis (arsphenamine)	40	F	28	14	109	2.2	
A. C.	Dermatitis venenata	38	F	34	18	79	2.2	
M. S.	Dermatalgia	48	M	34	16	70	4.2	
S.	Dermatophytosis	46	M	33	15	102	2.8	
H.	Dermatophytosis	40	F	39	20	88	3.9	
E. C. D.	Dermatophytosis	41	M	34	16	100	3.7	
C. F. B.	Dermographism	32	F	40	22	70	3.5	
P. T.	Neurodermatitis	31	F	30	14	116	2.2	
J. H. J.	Neurodermatitis	35	F	31	13	126	2.8	
M. L. B.	Neurodermatitis	54	F	37	17	98	3.1	
A. G.	Neurodermatitis	49	F	31	11	103	2.9	
E. A. R.	Dermatitis bullosa	39	F	34	16	87	3.8	
F. J. Mc.	Dermatitis eczematoid	62	M	35	20	133	4.2	
H. S. W.	Dermatitis eczematoid	26	F	30	15	97	3.3	
M. R.	Dermatitis eczematoid	..	M	35	14	90	6.0	
J. C.	Dermatitis exfoliativa	69	M	40	22	97	4.0	
F. A. S.	Dermatitis exfoliativa	39	F	27	13	95	3.3	
B.	Dermatitis exfoliativa	47	M	39	19	103	1.6	
W. S. B.	Dermatitis exfoliativa	55	F	35	16	144	5.4	After eczema; nephritis (retinal hemorrhages)
W. W.	Dermatitis exfoliativa	..	M	37	16	87	3.8	
H.	Dermatitis medicamentosa	40	M	34	17	65	3.3	
B.	Dermatitis seborrhoeica	34	F	32	16	98	4.1	
M. J.	Dermatitis seborrhoeica	36	F	28	11	101	2.8	
R. C. T.	Dermatitis seborrhoeica	57	M	42	23	94	2.4	
J. S.	Dermatitis seborrhoeica	66	F	45	24	100	4.6	
S. M	Dermatitis seborrhoeica	70	F	40	19	105	3.0	
C.	Dermatitis seborrhoeica	34	M	30	13	107	5.0	
J. S.	Dermatitis seborrhoeica	66	F	28	12	80	3.6	
H. S. C.	Dermatitis seborrhoeica	20	M	40	22	143	3.8	Later, dextrose 118
F. F. B.	Defluvium capillorum	28	M	42	19	100	4.8	
H. F.	Defluvium capillorum	37	F	32	16	98	2.0	
L. W.	Defluvium capillorum	37	F	29	11	87	3.4	
H. M.	Defluvium capillorum	35	F	28	10	110	2.2	
B.	Defluvium capillorum	60	F	30	13	95	2.4	
M.	Defluvium capillorum	44	M	33	17	107	3.6	
T. I. C.	Defluvium capillorum	58	F	31	14	97	3.7	
D. G.	Eczematoid dermatitis	52	M	35	12	102	2.6	
R.	Eczematoid dermatitis	61	F	35	11	112	2.3	
H. J.	Eczematoid dermatitis	45	F	28	13	100	2.9	
B. P.	Eczematoid dermatitis	48	F	..	..	82	2.5	
H.	Eczematoid dermatitis	38	F	40	19	75	2.2	
B.	Eczema circumscriptum	48	F	36	11	106	4.3	
D.	Eczema nuchae	60	F	36	17	119	5.8	Blood pressure, 290
W. H. L.	Furunculosis	37	M	37	18	66	2.6	
H. C. H.	Erythema induratum	51	M	31	15	99	5.0	Later, uric acid 6.0
L. F.	Erythema induratum, erythema elevatum	38	F	26	10	102	2.1	
F. K.	Erythema diutinum	41	M	32	15	89	2.7	
C.	Erythema multiforme	27	F	27	11	69	2.0	
K.	Acne	24	F	34	11	71	3.3	
I. B.	Acne	20	F	..	12	103	2.6	
F. H.	Acne	23	F	..	13	92	3.1	
G. M.	Acne	22	F	34	14	89	2.3	
S.	Acne	25	M	32	13	86	2.3	
W. E. G.	Acne	22	M	28	14	89	2.2	
H. W.	Acne	35	F	24	11	79	3.0	
F. C.	Acne	20	F	39	19	80	3.6	
S. H. D.	Acne	25	M	40	22	100	7.2	Slaty pigmentation of skin (possibly Addison's disease)

TABLE 2.—*Chemical Blood Studies in Miscellaneous Dermatoses—Continued*

Patient	Condition	Age	Sex	Nonprotein Nitrogen	Urea Nitrogen	Dextrose	Uric Acid	Comment
C. H.	Acne	16	M	..	..	70	4.2	
A. S.	Acne necrotica	45	M	30	15	142	3.3	Later, dextrose 96
W.	Acne rosacea	48	F	31	15	79	2.4	
B. O.	Acne rosacea	33	F	28	14	85	2.6	
F. R.	Acne rosacea	38	F	28	12	98	4.1	
H. B.	Acne rosacea	46	F	28	15	102	2.2	
K. O. H.	Acne rosacea	43	F	29	14	95	2.2	
C. D. G.	Acne rosacea	41	F	40	25	133	5.5	
	Acne rosacea	39	F	34	17	78	4.3	
L. W. F.	Acne rosacea	37	F	27	13	105	2.3	
D. C.	Acne rosacea	53	M	40	19	85	3.9	
G. E. J.	Acne rosacea	25	M	26	16	99	3.3	
S. G. F.	Acne rosacea	37	M	28	11	85	3.1	
J. H.	Acne hypertrophica nasi	75	M	36	19	133	4.4	
S. P.	Acneiform eruption	44	F	..	..	100	2.3	
B. P.	Alopecia areata	23	M	31	14	75	4.5	
H. S. H.	Alopecia areata	46	M	44	24	121	4.3	
J. D. B.	Alopecia prematura	20	M	30	14	110	5.0	
K.	Alopecia prematura	43	M	34	9	123	2.8	
A. B.	Alopecia prematura	35	F	38	19	102	2.5	
H. N. T.	Dermatalgia	40	F	30	13	88	3.6	
M. B. H.	Carbuncles	62	M	42	20	116	5.0	
L.	Chloasma	44	F	32	9	108	2.3	
J. K.	Dermatitis	45	M	37	18	93	1.9	
C. R.	Dermatitis	59	M	42	24	99	3.4	
J. M. N.	Dermatitis	58	F	35	17	127	2.5	Hemostatic; legs
H. L. W.	Dermatitis	50	F	37	17	85	3.0	
E. L.	Dermatitis	26	F	32	16	88	3.0	
M. G.	Dermatitis	25	F	..	..	80	2.4	
Dr. R. H.	Dermatitis	36	F	26	10	91	3.0	
H. K.	Dermatitis	35	M	26	14	97	2.7	
E. C.	Dermatitis	49	F	28	13	95	4.2	
M. N.	Dermatitis	40	M	37	20	100	6.4	
J. K. L.	Dermatitis	41	M	31	14	95	3.4	
E. K.	Dermatitis	57	F	47	26	106	3.7	
E.	Dermatitis	54	F	36	18	96	4.5	
R. S.	Acne rosacea	30	F	36	10	121	2.4	
I. W.	Acne rosacea	55	F	28	15	108	2.5	
F. M. L.	Acne rosacea	46	M	37	19	105	3.2	
J. O.	Acne rosacea	55	M	..	13	96	3.6	
A. E.	Acne rosacea	40	F	30	11	109	2.4	
W. J. B.	Acne rosacea	43	M	40	18	118	4.6	
C. E. C.	Acne rosacea	65	M	40	22	104	2.7	Also pruritus ani
C. F. C.	Acne rosacea	54	M	33	16	97	3.3	
L. B. S.	Acne rosacea pustulosa	57	F	46	12	123	2.7	
C. V.	Scabies	58	F	25	..	...	4.6	
J. S.	Scabies	62	M	31	15	77	2.4	
R.	Scabies	51	M	28	14	76	3.3	
H.	Scabies	..	M	..	..	40	2.0	
H.	Scabies	30	F	31	13	110	2.5	
W.	Seborrhea capitis	43	F	30	13	98	2.8	
C. T.	Tuberculosis cutis	60	F	37	18	103	5.0	
G. L.	Tinea cruris	20	M	37	13	96	3.3	
H. D.	Tinae cruris	48	M	34	..	...	...	
L. T.	Furunculosis	..	M	36	19	100	4.8	
H. B.	Furunculosis	32	M	45	21	97	3.7	
R. G.	Furunculosis	63	F	45	28	61	2.8	
J. B.	Carbuncles	43	M	28	11	74	2.9	
L. B.	Furunculosis	51	M	37	19	100	3.8	
J. B. T.	Folliculitis nuchae	44	F	32	15	110	3.2	
C.	Gangrenae cutis	31	..	38	17	109	2.6	
S.	Glossitis	..	F	40	22	81	1.1	
J. H.	Glossitis exfoliativa areata	35	M	36	16	110	5.0	
S. S.	Herpes zoster, general	70	M	46	24	90	3.3	
G. G. C.	Keratosis	60	F	34	14	95	4.8	
E. C. F.	Keratosis	71	M	42	20	143	...	
E. M. S.	Keratosis faciei	46	M	48	23	119	3.1	
S. B. B.	Keratosis faciei	72	M	39	17	97	3.9	
J. J. R.	Leukokeratosis buccalis	46	F	38	14	97	2.2	
J. S.	Leukoplakia	63	M	31	14	109	4.5	
R. B.	Lichen planus	52	F	34	17	84	2.4	Extensive
P. S.	Lichen planus	31	M	35	17	85	2.0	
N. S.	Lichen planus	60	M	42	22	75	4.2	
M. S.	Lichen planus	64	M	32	13	84	3.9	
L.	Lingual hemorrhages	53	F	..	14	...	2.8	

TABLE 2.—*Chemical Blood Studies in Miscellaneous Dermatoses—Continued*

Patient	Condition	Age	Sex	Nonprotein Nitrogen	Urea Nitrogen	Dextrose	Uric Acid	Comment
H. S.	Latent syphilis	55	M	42	19	121	4.3	
J. I. F.	Latent syphilis	74	M	50	27	94	5.5	
M. K.	Latent syphilis	48	M	35	18	96	4.4	
J. S.	Latent syphilis	61	M	35	18	100	4.6	
W. B.	Tertiary syphilis	61	M	36	16	116	2.8	
L.	Tertiary syphilis	46	F	35	17	106	4.0	
C. R.	Cerebrospinal syphilis	48	M	32	15	106	4.0	
J. O. S.	Cerebrospinal syphilis	50	M	26	12	86	4.3	
E. J.	Lupus erythematosus.	36	F	40	19	91	4.3	
M. S.	Lupus erythematosus.	25	F	35	16	102	4.0	
A. G.	Lupus erythematosus.	21	F	38	19	95	3.8	
C. S.	Lupus erythematosus.	40	M	..	11	...	4.0	
C.	Lupus erythematosus.	53	M	32	16	77	2.8	
L.	Lupus erythematosus.	52	F	23	11	85	2.3	
S. G. R.	Lupus erythematosus.	29	F	28	14	80	2.7	
J. F.	Lupus erythematosus.	26	M	23	11	59	5.0	
L. D.	Lupus erythematosus.	20	F	34	19	92	3.7	
W. G. O.	Lupus erythematosus.	35	M	35	18	91	3.1	
J. F. O.	Lupus erythematosus.	45	F	42	25	100	3.7	
A. L. O.	Lupus erythematosus.	40	F	39	22	97	4.5	
B. H.	Lupus erythematosus.	23	F	37	20	96	3.2	Acute type
Dr. H. W. B.	Nodular itching eruption	50	M	..	15	97	4.6	On scalp
M. S.	Mycosis fungoides	..	M	39	18	78	3.8	
B. F. K.	Mycosis fungoides	60	M	32	14	64 ?	5.0	
E. B.	Neurofibromatosis	47	F	34	17	95	3.8	
E. B.	Neurofibromatosis	47	F	39	19	87	3.2	
H. T.	Neurofibromatosis	21	M	33	19	84	3.1	
E. F.	Neurosis capitis	62	M	35	17	92	2.6	
J. C. F.	Neurosis cutis	62	F	20	..	118	2.7	
D.	Glossitis	..	..	..	..	...	...	
J. K. Y.	Parapsoriasis	60	M	31	15	117	4.7	
F. F.	Parapsoriasis	35	M	28	13	78	3.1	
J. K.	Pigmentationis	31	M	30	14	85	3.4	
M. S.	Progressive pigmentary disease	44	M	37	17	97	2.8	Cholesterol 202
J. S.	Progressive pigmentary dermatitis	64	M	34	17	129	4.5	
G. B. B.	Progressive pigmentary dermatitis	51	M	33	14	100	4.2	Cholesterol 300
W. H. L.	Epidermophytosis	..	F	28	13	92	3.3	
H. P.	Epidermophytosis	15	M	22	10	94	2.8	
O.	Prurigo nodularis	47	F	35	17	106	2.2	

RELATIONSHIP OF AGE

One might draw erroneous deductions if the age incidence of the different diseases was pronouncedly variant. For instance, the average age of the patients with acne was twenty-three years. The average content of blood uric acid of our patients with acne was 3.4 mg., and this includes one patient who had 7.2 mg. It would scarcely be fair, in view of the influence of age on uric acid, to compare this group, for instance, with the pruritus group in which the average age was fifty years. There was, however, relatively little difference, between the average ages of the patients in the pruritus, eczema and psoriasis groups. They were as follows: pruritus, fifty; eczema, forty-six, and psoriasis, forty-three years.

VALUE OF BLOOD CHEMISTRY IN PRESCRIBING DIETS

Even if we assumed what is not probable, that the figures which we found were more or less accidental and bore no relationship to eczema or pruritus, they would nevertheless be important in prescribing diets for patients. An excess of dextrose in the blood demands a

lowered carbohydrate intake. To no less an extent a distinct excess of uric acid requires a lowered purin diet, and an increase of nonprotein or urea nitrogen calls for a decreased abuminous content in the diet. Apart from the treatment necessary in view of the dermatosis under consideration, such a diet is necessary for the general welfare of the patient. But we are sure that the proper adaptation of the diet for patients suffering from various cutaneous diseases has accomplished much more than this. We feel convinced that this therapeutic measure has aided us greatly in effecting a disappearance of various eczematous and pruritic processes or in abbreviating their course.

In the great majority of cases it is relatively easy to lower the pathologic figures for blood nitrogen by close observance of a proper diet. This is sometimes less true of uric acid than of urea nitrogen. We have still much to learn about the cause of increased uric acid in the blood. In conclusion we may add that although we have examined the blood of only ten patients with acne we have not observed the hyperglycemia that a few writers have said exists in this condition.

TECHNIC EMPLOYED

The figures for the nonprotein nitrogen, urea nitrogen, dextrose and, in the earlier tests, for the uric acid were obtained by the methods of Folin and Wu.[1] The last two-thirds of the uric acid determinations were made by the method of one of the writers (H. B.[2]). This method has been thoroughly checked against other technics and has given accurate results.

CONCLUSIONS

1. Age exerts a distinct influence on the average nitrogenous content of the blood. From the third to the eighth decade there is a distinct and steady rise of nonprotein nitrogen, urea nitrogen and, particularly, uric acid.

2. Males exhibit more nonprotein nitrogen, urea nitrogen and uric acid in the blood than females.

3. In eczema and in pruritus, particularly of the generalized type, we found a perceptibly higher percentage of patients with an excess of nonprotein nitrogen, urea nitrogen and uric acid than in other dermatoses.

4. A study of the blood chemistry of patients with refractory dermatoses is of material aid in prescribing appropriate dietaries.

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1. Folin, O., and Wu: J. Biol. Chem. **38**:81, 1919.
2. Brown, H.: J. Biol. Chem. **68**:123, 1926.

PITYRIASIS FOLLICULORUM (DEMODEX) *

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Pityriasis folliculorum (*Demodex*) represents an apparent clinical entity no previous mention of which could be discovered in a careful search of the current textbooks on dermatology and of the dermatologic literature during the past twenty years. It is with some misgivings that the author attributes a pathogenic rôle to *Demodex folliculorum* in the face of an overwhelming opinion to the contrary due to the repeated finding of the parasite in the sebaceous ducts of many persons with normal skins. In fact, some authors have stated that the parasite occurs in the hair follicles or sebaceous ducts of nearly all adults.

By analogy with other disorders, however, this ubiquity of the demodex should not be considered an unanswerable argument against its assuming pathogenicity under certain circumstances. It is a familiar fact that although *Staphylococcus* can be cultured from apparently normal skins, it may at times cause disease. The common bacteria of the mouth occasionally assume virulent aspects, and certain common, ordinarily harmless intestinal parasites may, under special conditions, produce pathologic changes.

When one considers the size and appearance of the demodex as compared with *Acarus scabei,* one wonders how it is possible for such an organism to exist on or in the surface of the body without producing symptoms. According to Darier,[1] the parasite "is a worm-like acarus, measuring from 0.3 mm. to 0.4 mm., whose cephalothorax is provided with a mouth and four pairs of rudimentary feet; the abdomen resembles the finger of a glove and shows fine transverse striations. This parasite inhabits the sebaceous glands of the follicles, especially the mouths of the large sebaceous glands of the face, with its head directed inward; a large number, ten or twelve, may be found in the same follicle." *Acarus scabei* is somewhat shorter but more nearly round. The acarus, moreover, burrows into the epidermis for food or to deposit eggs, whereas the demodex is content to live on the fatty material secreted by the oil gland. Possibly the fact that the follicle is lubricated by the sebaceous material minimizes the friction which might otherwise be perceived from the crawling movements of the parasite.

Free drainage of the sebaceous glands, especially aided by daily washing with warm water and soap, would tend to keep the parasites reduced to a minimum number and to remove the waste products of their

* Submitted for publication, June 3, 1929.

1. Darier: Text-Book of Dermatology, translated by Pollitzer, Philadelphia, Lea & Febiger, 1920, ed. 2, p. 513.

alimentation which must be considerable if as many as ten or twelve parasites exist in a single follicle. It does not require a great stretch of the imagination to picture what would happen if deliberate measures were adopted whereby mechanical obstruction of the oil ducts were to take place. This is what happens as the result of the prevalent fad among seekers after beauty who have abandoned the use of soap and water for cleansing creams and skin foods. About the only things that are nourished by the skin foods are the demodex parasites. The job of obstructing the follicles is made complete by a liberal coating of heavy face powder. Cleansing creams may remove some of the surface dirt but for real mechanical cleansing they cannot compete with soap and water.

The eleven patients whose cases are herein reported were women, and only two of them used soap and water on their faces with any degree of regularity. All of these patients were seen within a period of less than four months, which leads one to believe that the condition is fairly prevalent, especially among the well-to-do classes.

REPORT OF CASES

Case 1.—Mrs. E. F., seen on Jan. 30, 1929, complained that her face "burned." On previous occasions, this patient had complained of a burning sensation and dryness of the face but there had been practically nothing to see, and the complaint was regarded as a neurotic manifestation. Soothing creams and lotions failed to give relief. On the aforementioned date, the patient insisted that something must be done. The condition had been present for more than a year and was gradually becoming worse. A burning sensation, dryness and a slight roughness which the patient characterized as resembling goose flesh constituted her symptoms. She said that she was unable to use soap and water on her face because of the dryness, and in fact had not employed soap oftener than once in two months for the past ten or fifteen years; she depended entirely on cleansing creams.

At a distance of 5 feet (152 cm.), practically no abnormality could be detected. On close inspection in a strong light, the face appeared to be somewhat dry, slightly redder than normal, and in certain areas, notably the sides of the forehead, chin and cheeks, also slightly on the front of the neck, there was a somewhat nutmeg grater-like or stippled appearance which was distinctly palpable and clearly visible, especially if the skin was stretched slightly. This effect was due to a white scaling at the base of the lanugo hairs which gave them a frosted appearance as though they had been sprayed with ethyl chloride. Under a hand lens the scales were seen to glisten, and they appeared to consist of a crystalline material.

Some of the scaling was picked off with a fine-pointed forceps and examined in its natural condition under the microscope. Large numbers of *Demodex folliculorum* were found, in some instances as many as five to a scale, and many of them showed active movements of the body and legs. After adding 40 per cent potassium hydroxide to the scales and heating the liquid gently, many more parasites were brought into view. It should be emphasized that these scales were picked off the surface of the skin and were not squeezed out of the follicles.

In spite of the patient's history of being unable to use soap, she was given liquid green soap to use daily and a rather vigorous ointment consisting of 2 Gm.

salicylic acid and 30 Gm. 5 per cent ammoniated mercury ointment to apply once daily. Five days later when the patient returned, the face was somewhat redder and more diffusely scaly, but she said that it felt much better. She was told to omit the ointment. Four days later, the skin still showed considerable erythema from the ointment, but a microscopic examination revealed a marked diminution in the number of parasites. After another four days the irritation had entirely subsided, and the patient said that her face felt much better than it had in months. There was a slight follicular scaling, however, which revealed numerous *Demodex* parasites, some actively moving. The patient was given an ointment commonly employed in the treatment for scabies: 2 Gm. beta naphthol, 4 Gm. sublimed sulphur, 15 Gm. balsam of Peru and 15 Gm. petrolatum.

Because the ointment caused a severe inflammation, it was used only once. When the patient returned in three days, however, the irritation from the ointment was subsiding. On February 21, or nine days after using the ointment, the condition was much improved; she was told to use no further medication except daily washing with soap and water, which she continued to do without the dire results which she feared, but with pronounced amelioration of her symptoms. About six weeks after using the ointment there was still slight follicular scaling in places, but examination of many fragments of material revealed only one demodex.

Case 2.—Mrs. G. C., seen also on Jan. 30, 1929, complained of dryness and roughness of the face of about four months' duration. Itching or burning was not mentioned. Several months previously, she had consulted a reputable dermatologist who had prescribed a soothing ointment, without benefit. Until the last month or two, she had not used soap on her face for many years.

Close inspection of the face revealed a diffusely rough, dry appearance with the characteristic nutmeg grater stippling, consisting of fine white scaling about each lanugo hair; this was especially noticeable on the forehead, across the middle portion of the nose, the sides of the cheeks and the chin. There was an inconspicuous flush. Microscopic examination of the scales showed *Demodex* in considerable numbers.

Daily vigorous cleansing with warm water and soap and two series of applications of the salicylic acid and ammoniated mercury ointment resulted in a complete symptomatic cure as observed more than three months later. One subsequent microscopic examination on February 19 showed a pronounced decrease in the number of organisms.

Case 3.—Mrs. R. S., who had been under treatment several months for psoriasis, called attention, on Feb. 7, 1929, to a dryness, irritation and slight scaling of the face which had been present for several years. She had not used soap on her face for over thirty years.

The face was slightly red and rough. About the bases of the lanugo hairs where they emerged from the skin could be seen the characteristic frosted white scaling, most noticeable on the sides of the forehead and on the chin. *Demodex* was found in moderate numbers. The patient was given a sulphur ointment used for scabies and was advised to wash with soap and water. The ointment provoked temporary irritation, but when she was observed two weeks later, the condition had improved. The patient's return to the east interrupted further observation.

Case 4.—Mrs. E. S., who was seen on Feb. 11, 1929, complained of irritation and dryness of the face of several years' duration. For over ten years, she had not used soap oftener than once or twice a week. The face was dry, and slightly rough, and showed follicular scaling, especially on the forehead, the medial portion

of the cheeks and on the chin. Microscopically, *Demodex folliculorum* was seen in small numbers. A compound sulphur ointment was prescribed to be used on alternate nights for three applications. The patient has not reported since.

CASE 5.—Mrs. A. D., seen on Feb. 13, 1929, complained that for three years her face had been dry, hot and irritated. For the past year she had used no soap. The present rather acute outbreak was of two weeks' duration. The patient presented a diffusely red, scaly condition of the sides of the neck, forehead and chin. In addition, a characteristic follicular scaling was noted on the sides of the forehead and nose and beneath the chin and jaws. *Demodex* was easily demonstrated in scales from the forehead and nose. Because of an apparently acute toxic dermatitis superimposed on the more chronic follicular pityriasis, systemic measures and four fractional doses of x-rays were employed. The acute condition responded satisfactorily, but the patient did not return for treatment.

CASE 6.—Miss A. M., who was first seen on Feb. 23, 1929, had been troubled with an irritation of the face for a year. It had become much worse during the past three months. During the past year she had used soap irregularly, sometimes omitting it for a month at a time. The skin was of an extremely delicate, Irish type; the face, especially the central portion, was diffusely flushed, oily and somewhat rough, presenting typical follicular scaling, especially on the cheeks, nose, chin, and sides of the forehead. *Demodex folliculorum* was readily demonstrated in follicular scales. Because of the extreme delicacy of the skin and a suggestion of acne rosacea, milder applications were tried first without success. Finally, on April 8, the scabies ointment mentioned in the report of case 1 was prescribed. The patient made two applications and suffered some irritation, but about one month later she reported by telephone that her face was much better. On May 21, the face looked practically normal, and the feeling of irritation had practically subsided. A few parasites were seen microscopically.

CASE 7.—Mrs. E. H. was first seen on Nov. 28, 1928, complaining of a burning, irritated condition of the face which had lasted about two years. A diagnosis of acne rosacea was made, and appropriate applications were prescribed. The patient returned on April 17, 1929, unimproved. At this visit, careful inspection revealed typical follicular scaling, and the microscope showed *Demodex*. On questioning, the patient admitted that she had used soap infrequently. The sulphur ointment used in case 1 was prescribed, but the patient did not report for further observation.

CASE 8.—Mrs. S. C. was seen on April 1, 1929, for a burning condition of the face which had been present intermittently for about four years. She had used no soap on her face for five years. A characteristic frosted, follicular scaling was seen, especially on the forehead. *Demodex folliculorum* was found in considerable numbers. The sulphur ointment was used for three nights and produced some irritation which, however, quickly subsided. The burning sensation and the follicular stippling had disappeared by April 22. On May 3, the patient reported that her face had remained well. One subsequent microscopic examination revealed a marked diminution in the number of the parasites.

CASE 9.—Mrs. F. C. was seen on April 12, 1929, complaining of a prickling, burning sensation of the face of about one week's duration. She said that she had always used soap and water daily, although she used cold cream freely. The skin was slightly red and dry, especially on the upper lip and the sides of the nose. Follicular scaling was readily seen, which under the microscope revealed *Demodex*. The sulphur ointment was used for two nights with a moderate degree of reaction. The patient was seen only once more, three days later, when the skin was still red from the ointment.

CASE 10.—Miss G. E. was seen on May 1, 1929, primarily for psoriasis. Careful inspection showed the typical follicular stippling of the face. She had been unaware of this condition until about three weeks before, when she noticed some irritation of the face. She had always used soap daily. Microscopic examination showed the presence of large numbers of parasites, as many as sixteen in one field. She has not yet had treatment for *Demodex*.

CASE 11.—Miss G. K., who was seen on May 10, 1929, had been troubled for four years with an intermittent irritation of the face, which had never entirely disappeared. It had become much worse during the past year. A dermatologist whom she had previously consulted prescribed bland ointments and lotions without any beneficial effect. She had used no soap on her face for two years until six weeks before. The eruption was characteristic, with slight flushing, dryness and follicular frosted scaling. Large numbers of *Demodex* were found microscopically. The compound sulphur ointment was prescribed. Four days later the patient returned, with the face temporarily redder and more scaly from the ointment. Only one live demodex was seen under the microscope, and its movements were feeble. More organisms were seen, however, in a potassium hydroxide preparation. On May 28, 1929, the skin appeared normal, and no parasites were found.

REVIEW OF THE LITERATURE

No definite mention can be found of the symptom complex reported in the foregoing cases; namely, a chronic, dry, slightly erythematous irritation of the face with follicular scaling and sensations of burning, occurring in women, particularly in those who give a history of not using soap, the constant finding in the scales of *Demodex folliculorum* and clinical improvement in all cases which could be followed, after the daily use of soap and water and the employment of vigorous antiparasitic remedies. Sufficient time has not elapsed to enable one to draw conclusions as to the permanence of the results. According to Lawrence,[2] the parasite is attacked with difficulty because of the depth to which it penetrates the oil glands. He advocates sexually sterilizing the parasites by means of x-rays to prevent them from multiplying; he feels that this is well within the range of practical therapeutic dosage.

Lawrence[3] stated that he was doubtful about the innocence of *Demodex folliculorum*. He mentions several cutaneous disorders in which clinical cure and the microscopic disappearance of the parasite occur simultaneously following antiparasitic remedies. He mentions certain types of impetigo, a case resembling lupus erythematosus, cases resembling pityriasis alba and several instances of rosacea. A closely related parasite in dogs causes "red mange," and in many cases is practically incurable. Lewandowsky[4] also reported a case of *Demodex* impetigo.

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2. Lawrence: Further Observations upon the Pathogenicity of Demodex (Owen) in the Human Being and of Other Parasites Producing Skin Diseases, M. J. Australia **2**:419 (Nov. 12) 1921.

3. Lawrence: The Pathogenicity of the Demodex (Owen) in the Human Being, M. J. Australia **2**:39 (July 16) 1921.

4. Lewandowsky: A Case of Impetigo-like Skin Disease in Man Caused by Demodex Follicularis Canis, Deutsche med. Wchnschr. **33**:801, 1907.

Chambers and Somerset [5] supported Borrel, Gastinel and Gorescu [6] in the suggestion that *Demodex folliculorum* may have some bearing on cancer of the breast, although no definite evidence exists to prove or disprove the theory.

Kaufman-Wolf [7] noted a definite association between *Demodex* and rosacea in women; she quoted Allen as presenting a case in a woman in whom lesions resembling molluscum contagiosum were found to contain large numbers of the parasites.

Darier [8] suspected that *Demodex* had contributed "an irritative influence" in a case of lichen spinulosus.

De Amicis, Majocchi and Dubreuilh [9] reported instances of pigmentation of the face similar to tinea versicolor. The condition reported by Dubreuilh was the most similar to that described in this paper; his patient showed "slight, but scarcely perceptible, follicular prominence, due to minute corneus projections from the orifices, associated with, in one or two instances, trifling scurfiness." De Amicis reported successful treatment by washing with soft soap. Dubreuilh failed to obtain any benefit from stimulating parasiticide applications.

SUMMARY

1. Eleven cases observed during the past four months presented a fairly uniform symptom-complex, which is tentatively designated as pityriasis folliculorum (*Demodex*).

2. All these patients were women, and all but two used soap on the face infrequently or not at all.

3. The symptom-complex consists in a sensation of burning or irritation of the face, together with dryness and a characteristic roughness due to the accumulation of a white, frosted-looking scaliness about the base of the lanugo hair giving rise to a stippled or nutmeg grater-like appearance.

4. In all patients in whom the subsequent course could be followed, the vigorous use of soap and water and a strong parasiticide ointment was followed by clinical improvement and by a diminution in the number of parasites found on microscopic examination.

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5. Chambers and Somerset: Breast Disease and the Demodex Folliculorum, Lancet **208**:172 (Jan. 24) 1925.

6. Borrel, Gastinel and Gorescu: Acariens et Cancers, Ann. de l'Inst. Pasteur **23**:97, 1909.

7. Kaufman-Wolf: Concerning the Usual Presence of Demodex Folliculorum in the Pustules of Acne Rosacea, Dermat. Wchnschr. **81**:1095 (Jan. 25) 1925.

8. Darier: Text Book of Dermatology, translated by Pollitzer, Philadelphia, Lea & Febiger, 1920, ed. 2, p. 513.

9. De Amicis, Majocchi and Dubreuilh, quoted by Stelwagon and Gaskill: Diseases of the Skin, Philadelphia, W. B. Saunders Company, 1921, ed. 9, p. 1258.

TRYPARSAMIDE IN ACUTE PEMPHIGUS*

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The treatment for acute pemphigus by the use of an arsenical compound, iron cacodylate or sodium arsenate, given intravenously, and coagulen, given subcutaneously, devised by Dr. Robert H. Davis and the late Dr. William D. Davis, was the first advance ever made in the treatment for this disease, and has yielded a large proportion of recoveries in what had until then been an all but hopeless condition. This has been confirmed by reports from many parts of the country.

Any therapeutic procedure may fail at times. It was such a failure (case 1) that led me to essay a modification of the Davis treatment. Meeting with an unhoped for measure of success, I employed the modified method in four other cases.

CASE 1.—This case has already been reported in detail.[1] Briefly, it concerned a woman, aged 32, of previous excellent health. Three weeks before consulting me, in January, 1927, she had developed itchy spots on the face and arms. After five days, vesicles, thought to be chickenpox, had appeared on those sites and on the tongue and palate. When I first saw the patient, there were many blebs the size of a hazelnut on the face, neck, shoulders and arms, and a few on the back, abdomen, thighs and vulva.

She was admitted to St. Mary's Infirmary and was at once placed on the Davis treatment, which was rigorously followed. The blebs, however, continued to grow larger and more plentiful, so that on the eleventh day of observation they were from 2 to 3 cm. in diameter, and so thickly spread over the arms, forearms, thighs and buttocks as to be coherent in many places. The temperature rose to 105 F. The white cell count was 14,500, and the eosinophils numbered 15 per cent. The eruption continued to spread until virtually the entire surface was covered with bullae or with raw patches twice the width of the palm or larger, where bullae had coalesced and broken. The nasal passages were painful and bled; the mouth showed large denuded patches, making feeding difficult. She had become pale and was rapidly becoming emaciated. Her condition was pitiable in the extreme. In view of the fact that she had grown steadily and rapidly worse in spite of the measures employed, it was decided to try the effect of another arsenical compound. Tryparsamide, a pentavalent compound derived from atoxyl, suggested itself on account of its low relative toxicity and its value in raising tissue resis-

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1. Grindon, Joseph: Davis Treatment and Tryparsamid in Acute Pemphigus, M. Clin. North America **11**:507 (Sept.) 1927.

tance, as seems to be shown by its good effects in late neurosyphilis. As the kidneys and eyegrounds were apparently normal, 2 Gm. of tryparsamide was introduced intravenously. This dose was given four times at intervals of one week, and was followed by two doses of 1 Gm. each. Improvement was noticeable within a few days after the first injection. Only a few new lesions appeared, while the old ones rapidly dried. The temperature soon fell to normal. At the end of five weeks, the surface was clear save for the appearance of a small vesicle every day or two. The patient left the hospital after eleven weeks, still showing an occasional vesicle the size of a pinhead. She continued well (June, 1929) save for occasional minute intra-oral lesions. At the time of writing she was on a reducing diet, her weight having risen to 179 pounds (81.2 Kg.).

Case 2.—A white boy, aged 5 years, was admitted to the Isolation Hospital on June 25, 1927, with a condition that had begun with lesions on the abdomen, which the mother attributed to a phenol solution. Soon after, a few small blebs appeared on the left arm. There was pain in the left ear. Dr. Lohr and Dr. Garrison furnished the following report:

On admission, the patient's temperature was 101.4 F. There were small bullae about 3 mm. in diameter about the lower lids of both eyes and the conjunctivae were much inflamed. Many blebs were seen over the left ear and side of the face, and some on the neck and back. Over the lower part of the abdomen, the genitals and the inner aspects of the thighs were numerous bullae, with one on the right knee. The results of the physical examination were otherwise negative. Five days later, the blebs had increased considerably in number. The patient was then given tryparsamide, 0.33 Gm., intravenously. The highest temperature thereafter recorded was 99.4 F. Within a week, many old lesions healed. No new ones appeared. Later, weekly doses of 0.66 Gm. were given. The patient vomited once after the second dose but had no other reaction. The white cell count was 14,000. The fluid aspirated from the intact bullae yielded a pure culture of *Staphylococcus aureus*. At the end of a month, the patient was practically well except for a denuded area on the left arm and one over the lower part of the abdomen. By August 20, all of the lesions were healed. At the St. Louis meeting of the Dermatological Conference of the Mississippi Valley the patient was shown in November, 1927. Since that time the patient has continued well.

Case 3.—A negro, aged 35, entered City Hospital No. 2 on March 16, 1928, with a condition that had existed for two weeks, beginning with lesions in the mouth and throat, and headache. Two days later he developed fever, edema of the feet and prostration, soon followed by pain in the muscles and joints. Blebs appeared on the fourth day. These tended to heal at the center and spread at the edge. On entry there were relatively few bullae, which were generally distributed. The blood count revealed: leukocytes, 20,000; eosinophils, 30 per cent. The mouth and lips, as well as the throat, were so sore that feeding was impossible without prior swabbing with cocaine. The patient was given tryparsamide, 2 Gm., intravenously once a week, with coagulen, 1.5 cc., on alternate days. Improvement was evident from the first, no new lesions appearing after the third day. Recovery was rapid, and after two weeks the lesions were practically healed. The patient was discharged on the twenty-sixth day, but continued to report at the hospital.

Case 4.—About March 1, 1928, a white woman, aged about 40, sustained a wound of the scalp, which suppurated. On March 26, she developed bullae on the neck which gradually spread over the body and to the buccal mucosae and palate. There were large flat pustules on the back of the neck and numerous bullae, varying in size from that of a small pea to that of a large pea, in the axillae and on

the arms. Over the general surface was a patchy erythema, in places crowded with papules and vesicles. There was a discharge of pus and blood from the nose and superficial ulceration of the palpebral conjunctivae. The patient was admitted to St. Mary's Infirmary on April 2. The white blood count was 6,200, the red blood count 4,450,000, and hemoglobin (Newcomer) 80 per cent; the differential count showed: polymorphonuclears, 71; lymphocytes, 23; large mononuclears, 1 and eosinophils, 5 per cent. The nonprotein nitrogen and blood sugar were normal. The Wassermann reaction was negative. The phenolsulphonphthalein test gave a return of 25 per cent in the first hour and 22 per cent in the second hour. Urinalysis gave negative results. The patient seemed to improve for a day or two, but on April 6 a number of new lesions appeared. As the kidneys and fundi of the eyes were found apparently normal, she was given 2 Gm. of tryparsamide intravenously once a week, and 1.5 cc. of coagulen subcutaneously on alternate days. Cultures from the blebs and throat yielded staphylococci. On April 11, the blood count showed: white cells, 7,800; polymorphonuclears, 54 per cent; lymphocytes, 33 per cent; large mononuclears, 4 per cent, and eosinophils, 9 per cent.

For a time the lesions continued to increase in size and number, breaking and coalescing into huge raw surfaces, which for a time extended continuously over the scapular region and both buttocks, so that for weeks the patient lay on her chest. Improvement then slowly set in, but as there had been some albuminuria and occasional casts, with swelling of the feet and ankles for several weeks, the weekly injections of tryparsamide were stopped on June 7, and were not resumed until July 7, after the albuminuria and casts had been absent for a number of days. Ten doses had been administered.

Improvement continued, until by June 21 the lesions of the mouth had healed and there was only one new bulla, on the right ankle.

On August 13, four months after commencing treatment, the patient called at my office. There were one new bulla on the wrist and some minute lesions on the face and neck. A week later, she returned to her home in a neighboring town. During September and October, there were many new small bullae, and by November 1 there were many fresh lesions which confined the patient to bed. She was again given tryparsamide, and again the conditions improved, so that by November 30 she was able to go to the city. There were then numerous crusts from the small lesions, but she protested that she felt well. For the next two months there was only a rare bleb. In February, she contracted a bilateral lobar pneumonia, of which she died on March 5.

The four cases just detailed were of an acute type. The next one pursued a chronic course.

CASE 5.—A white man, aged 58, Jewish, was first seen on Feb. 24, 1928, at which time my diagnosis was erythema multiforme bullosum. Dr. R. H. Davis, who was present, concurred. The fact that the patient had been having recurrent raw patches in his mouth for nine months was not elicited until later. Lesions had existed for several months, and consisted of broad erythematous patches of somewhat urticarial aspect bearing here and there blebs of varying sizes. The patches were generally distributed, but were most numerous over the hips, thighs and legs. The patient was admitted on the same day to St. Mary's Hospital, where after some days the true nature of the condition became apparent.

The lesions rapidly increased in size and number, nearly covering the body with bullae and huge denuded surfaces. The blisters in the mouth recurred and

became a troublesome feature; nevertheless, the patient's appetite remained fair, as did his general condition. At no time was there a marked rise in temperature. From the first, tryparsamide was administered intravenously in doses of 2 Gm. once a week and coagulen in doses of 1.5 cc., every alternate day.

The treatment was well borne, but the patient grew gradually worse. At the end of six weeks, the use of tryparsamide was discontinued, and the original Davis treatment was substituted. This was continued during the remainder of the patient's stay in the hospital (three weeks), but with no apparent benefit. He returned to his home in another city on May 3. Reports received since then stated that at times practically the entire body had been involved, including the scalp, face and neck. Again, an entire extremity had been nearly free for a time. The blebs were smaller than formerly, being 2 cm. or less in diameter. The knees and ankles had been swollen and painful for a week or two at a time. There had been several attacks of diarrhea, and hoarseness, probably due to laryngeal pemphigus, was present. Emaciation had progressed. He received two or three injections of iron cacodylate per week. He died on March 17, twenty-two months after the appearance of the lesions in the mouth.

When first seen the red blood count was 4,060,000 and the white cell count, 14,700. The differential count revealed: polymorphonuclears, 60 per cent; small lymphocytes, 18 per cent; large lymphocytes, 12 per cent and eosinophils, 10 per cent. A month later the blood count was: red cells, 4,000,000; white cells, 13,000; polymorphonuclears, 52 per cent. The lymphocyte count had not changed much, and the eosinophils had risen to 22 per cent. Bacteriologic examination of scrapings from the lesions on the mouth and body showed *Staphylococcus aureus* and *Streptococcus hemolyticus*. No yeasts or fungi were found (report of Dr. O. Garcia).

Dr. Collier reported as follows: "Sections of a bulla near the knee show a subacute bacterial dermatitis. The underlying epidermis shows moderate thickening, a mild polymorphonuclear infiltration and intradermal miliary abscesses. At most sites there is a large number of eosinophils. Staining for bacteria shows gram-positive and gram-negative diplococci. There are no acid-fast organisms, branching forms of bacilli or yeasts."

Dr. Oliver S. Ormsby communicated the following report of the case of a patient with pemphigus whom he treated with tryparsamide:

The patient, aged 57, had a condition of one year's duration. He was seen in September, 1926, at which time the lesions were generally distributed, including many in the mouth and some on the genital mucosa. Treatment with iron cacodylate and coagulen, according to the Davis method, was employed for fifteen months, with much improvement. In December, a series of injections of tryparsamide was instituted. These were given at weekly intervals, until six were employed. Shortly after this the pemphigus lesions disappeared, and there has been no recurrence up to the present time (sixteen months).

In a case reported by Dr. R. H. Davis[2] (case 7), the condition grew progressively worse under treatment with three weekly doses of tryparsamide, but later the patient made a clinical recovery with the use of iron cacodylate and coagulen. It is to be noted that the dose of tryparsamide employed was 1 instead of 2 Gm. as in my adult cases.

2. Davis, R. H.: Treatment for Pemphigus: Further Report, Arch. Dermat. & Syph. **19**:451 (March) 1929.

COMMENT

The cases cited are too few to permit of the evaluation of this modification of the Davis treatment as compared with the original method. It may be that one arsenical compound is better fitted to one type of the disease, and another to another. The results obtained with tryparsamide were gratifying in the acute cases and in one chronic case (Ormsby), but were disappointing in the other chronic case.

SUMMARY

1. Four cases of acute pemphigus are reported, in which the patients were successfully treated with tryparsamide and coagulen; also two cases of chronic pemphigus in which the same treatment was used, with one success (Dr. Ormsby's case) and one failure.

2. Three of the four patients with acute cases showed prompt and rapid improvement, with apparent cure. In the fourth, improvement was delayed, and the lesions did not completely disappear. There was a remission, which on renewal of the treatment again showed improvement falling just short of complete clinical cure.

3. A decision as to the comparative value of the use of tryparsamide in pemphigus must await further trial.

ABSTRACT OF DISCUSSION

DR. MCEWEN, Chicago: I think that Dr. Grindon is to be congratulated on the opportunity he has had for making this test with a comparatively new arsenical compound, and also for his success in the use of the modified Davis treatment. He is to be commended for reporting his results for the benefit of others. One might almost conclude from what he has said that tryparsamide is an arsenical drug that can be used in place of iron cacodylate in pemphigus of the acute type, and that in the chronic type its substitution value is not proved. This, of course, constitutes a step in advance in the study of the Davis treatment, but it still leaves the mystery of the success of that treatment unsolved.

The problem gives rise to a number of questions: Is arsenic the essential element in the Davis medication, the really curative factor? If it is, quantity seemingly is not important, for the total arsenic content of the iron cacodylate administered is not large. Is iron the necessary element? Apparently not. Dr. Grindon's paper shows that good results may follow the use of an arsenical compound without iron. What is accomplished by the coagulen? Would it be possible to get results by treating patients with pemphigus with iron cacodylate or tryparsamide without the associated use of coagulen? And what would be the outcome if coagulen were used alone without the arsenic? It may take courage to omit the arsenic and to trust to the other medicament alone, but how else can the experimental determination of curative factors of the Davis treatment be made? In this connection I call to mind a fulminating case in which widespread bullae of small size were observed to absorb and flatten within twelve hours after the injection of one ampule of coagulen, no arsenic having been administered.

It seems to me that the real solution of the treatment for pemphigus lies in the discovery of the cause of the condition. It is apparently an infection, and

when the etiologic factors have been definitely established the reason why the Davis treatment has proved successful in so many cases will become apparent and the way will be cleared for further advancement in the therapy of this serious disorder.

DR. CANNON, New York: Those of us who are associated with large municipal hospitals in which we see a relatively large number of cases of pemphigus should be grateful to Dr. Grindon for suggesting anything that may offer a cure for this disease. Of about forty-five patients with acute pemphigus whom I have treated during the past several years, I recall only two who have been free from lesions as long as from three and one-half to seven years; although two other patients have been free from skin lesions for several years, they have continued to have involvement of the mucous membranes of the mouth. We have used the Davis treatment, arsenic, arsphenamine, blood transfusions, mercurochrome-220 soluble and various local remedies, and I am certain that we have had better results from thorough alkalinization than from any other kind of medication. This we accomplish by giving sodium bicarbonate, sodium citrate, sodium thiosulphate and sodium salicylate. After the patient has been on this form of treatment for two or three weeks, we discontinue it for a short while and give small doses of neoarsphenamine, from 0.2 to 0.3 Gm., at weekly intervals, then return to the alkalis. In this way we have seen remarkable improvements, but the patient has a return of the lesions within from a few weeks to a few months.

DR. MACKEE, New York: I am glad to have this report on the use of sulpharsphenamine. The more I see of pemphigus, the more I am inclined to divide the cases into two types—the fatal ones and those that are not fatal. I really do not mean to be facetious. What I mean is that there is an acute type that nothing can cure; there is also a type with an indefinite course which, no matter how it may start, whether insidiously or acutely, I doubt can be controlled much by therapeutic efforts, except perhaps by hospitalization, diet, etc. I think that in some instances arsenic may be of assistance. We all know that the disease acts differently in different cases, a question probably of either localized or generalized immunity. A patient may have lesions of the mouth for a year or two and none of the body. We cannot diagnose, but we suspect pemphigus. Perhaps a year or two later a generalized eruption appears, perhaps followed by death and perhaps not. In other instances, there is an acute, generalized eruption that will dwindle into the chronic type. While I always use arsenic and other recommended remedies, I am inclined to believe that Nature does more than we do, except by hospitalization, proper nursing, proper diet, etc. In other words, pemphigus is a problem of immunity and we endeavor to keep the patient alive until immunity develops.

DR. GRINDON, St. Louis: As to Dr. McEwen's question as to which agent, coagulen or arsenic, is effective, I do not know, but it is my belief that it is the arsenic. I did not feel that I had the right to make what would have been an interesting experiment in giving one drug alone. As both were recommended, I felt that the patient should have the benefit of all the possibilities.

Dr. McEwen suggests that still other arsenicals would probably be of use. I felt that arsacetin held out some promise, but tryparsamide did so well that I have not so far felt inclined to change. As Dr. McEwen said, we shall be better able to judge which is the best treatment when we know the cause. I do not believe that there is a single cause of pemphigus, but that it is due to a special reaction of the skin to a variety of organisms. In my cases, staphylococcus was found. Others have reported other organisms.

Dr. Cannon spoke of the use of neoarsphenamine. I am interested to know that small doses were helpful. Neoarsphenamine has been used, but so far as I hitherto knew, with little success. On analogical grounds we would expect benefit from pentavalent arsenicals in lesions affecting the ectoderm and from the trivalent compounds in lesions of mesodermic tissues.

There is no doubt, as Dr. MacKee brought out, of the benefit of hospitalization. However, my former patients were hospitalized and all died, but since I have done something besides hospitalizing them, five of six patients, including Dr. Ormsby's, have remained alive. This condition has hitherto been so uniformly fatal that even the suggestion of benefit that can be drawn from six cases is something that I did not have the right to withhold. I would not speak of cure in any of the cases, but of symptomatic cure, as shown by the fact that three of the patients are known to be well after sixteen, eighteen and twenty-four months.

THE QUESTION OF ARSPHENAMINE-RESISTANT SYPHILIS *

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Considerable interest has recently been aroused, especially in Germany, as to the possibility that the prolonged use of arsphenamine products in civilized countries has appreciably altered the course of syphilitic infection. It is argued pro and con whether late syphilis, especially of the cardiovascular apparatus and of the nervous system, is increasing or decreasing, and whether the classic time relationships of the prearsphenamine era, between infection and the appearance of late manifestations, have been altered. A study of the literature convinces one that no definite statements can as yet be made as to a possible increase of aortic syphilis or neurosyphilis, but it does seem clear that the time interval between infection and the appearance of these or other late manifestations of syphilis has been shortened.

In addition, there is much speculation as to the production of an arsphenamine-resistant strain of *Spirochaeta pallida* with a consequent increase in so-called arsphenamine-resistant syphilis. In certain German clinics, the impression prevails that in early syphilis, especially, treatment now is not as efficacious as in former years. The most important paper on this subject is that of Silberstein [1] from Scholtz' clinic in Königsberg. This clinic has been utilizing essentially the same scheme of treatment for early syphilis since 1911. Silberstein found that it has been much more difficult to reduce the Wassermann reaction of the blood to negative in recent years than formerly, as shown in table 1, and further that relapses of a secondary type were more frequent in the period from 1921 to 1922 than in that from 1911 to 1916 (47.5 per cent as compared with 16 per cent). It should be pointed out that the number of cases from which he drew conclusions for his second period (1921 to 1922) is too small for statistical accuracy. He also gathered from the literature reports of thirty-nine cases in which arsphenamine failed to heal the patients' original (secondary) lesions, or in which new and resistant lesions appeared during or shortly after a course of arsphenamine.

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* From the Syphilis Division of the Medical Clinic, the Johns Hopkins Hospital.

1. Silberstein, S.: Zur Frage der salvarsanresistenten Lues, Arch. f. Dermat. u. Syph. **147**:116, 1924.

The whole subject of arsphenamine-resistant syphilis was reviewed by Nathan,[2] who surveyed the literature thoroughly. He was convinced, as were many other German and some French observers, that it is becoming increasingly difficult to influence favorably the course of early syphilis, and stated that in order to accomplish the same results as in the early days of arsphenamine, it had been necessary in many clinics to increase the individual dosage and the length of courses and to shorten the interval of rest between courses (the system of intermittent treatment). He seemed also convinced that cases of syphilis definitely resistant to arsphenamine are more frequent in recent years, though no exact figures are available on this point. He stated, as did others, that if this situation is actually the case, it may be due to an alteration in the properties of the drug, in the response of the causative organism to treatment or in the patient. All German authors have united in feeling that the drug cannot be incriminated and that its

The Response of the Wassermann Reaction of the Blood to Treatment in the Scholtz Clinic, Königsberg (Silberstein)*

Time Period	Total Number of Cases	Percentage Which Became Wassermann Negative After		
		1 Course	2 Courses	3 Courses
1911–1916	236	85	97	100
1921–1922	60	23	65	76

* Silberstein: Arch. Dermat. & Syph. **147**: 116, 1924.

therapeutic value is as great now as formerly. This opinion is based on animal experimentation as to the toxicity and therapeutic activity of the arsphenamines, and on the fact that in most cases of early syphilis, lesions heal as promptly as in former years.

There is much difference of opinion as to the question of alteration of the biologic properties of the spirochete. Some workers believe that the universality of arsphenamine treatment in civilized countries is gradually producing a strain or strains of arsphenamine-resistant organisms. Others, lacking definite proof of this, hold the question sub judice. Resistance to arsphenamine is not a property of the organism capable of demonstration in the experimental animal; that is, several workers, notably Hoffmann and Armuzzi [3] and Nothaas [4] inoculated rabbits with organisms recovered from arsphenamine-resistant patients and were unable to demonstrate any difference from

2. Nathan, E.: Ueber salvarsanresistente Lues, Klin. Wchnschr. **6**:2147, 1927.

3. Hoffmann, E., and Armuzzi, G.: Experimentelle Untersuchungen über salvarsanresistente Syphilispirochäten, Deutsche med. Wchnschr. **53**:51, 1927.

4. Nothaas, R.: Experimentelle Untersuchungen über salvarsanresistente Lues, München. med. Wchnschr. **74**:761, 1927.

other recently isolated strains either in the course of the infection that resulted or in its response to treatment. Nor has it proved possible to demonstrate any high degree of arsphenamine fastness in experimental syphilis in rabbits by treating the animals repeatedly with subcurative doses, though Klauder's [5] experiments are suggestive.

It has been repeatedly pointed out, especially by Jessner,[6] that some clinical indication as to the question of drug-fast strains of organisms might be gained by a study of two or more persons infected with the same strain, as, for example, husband and wife, the infection in one of them running an arsphenamine-resistant course. Unfortunately, the available cases are as yet too few, and the evidence too conflicting, to be of value.

It is further suggested that, in Germany at least, the apparent increase in arsphenamine-resistant syphilis may be due to an alteration in the reaction of the subject to infection because of the lowered state of nutrition (and presumably also of resistance to all infections) produced by the results of the blockade during the World War. That this explanation may be true is suggested by the facts presently to be detailed, indicating that in the United States no such increase in arsphenamine-resistant syphilis is obvious.

Interesting sidelights on this question, amply confirmed by our own and other American experience, were mentioned by Jessner.[6] He pointed out that arsphenamine-resistant skin rashes are frequently psoriasiform, that seborrheic-like nodular lesions on the face are common and that a serpiginous arrangement of individual lesions is often observed. He further stated that in his own experience, as well as in that of others, patients with arsphenamine-resistant syphilis often react badly to arsphenamine; that is, annoying or moderately severe reactions of all types seem more frequent in them than in other patients. He suggested on this basis that the state of resistance to arsphenamine may be due to the fact that such patients metabolize arsphenamine in a different fashion from normal persons, therefore reducing its therapeutic activity.

Interestingly enough, moreover, in clinical practice, resistance to arsphenamine is often specific for a single member of the arsphenamine group, that is, lesions which refuse to heal under treatment with neoarsphenamine will often heal promptly after the administration of old arsphenamine or silver arsphenamine, or vice versa; and even if this is not the case, the phenomenon of resistance to arsphenamine is almost always group specific; that is, if lesions fail to heal with

5. Klauder, J. V.: The Experimental Production of an Arsenic Resistant Strain of Spirochaeta Pallida in Rabbits, Arch. Dermat. & Syph. **9**:446 (April) 1924.

6. Jessner, M.: Ueber salvarsanresistente Lues, Med. Klin. **19**:857, 1923.

any of the arsphenamine products, they usually clear up promptly after the administration of bismuth or mercury. It is exceedingly rare to find a patient whose lesions are resistant to all three drugs. These facts are also confirmed by our own experience.

The Wassermann reaction of the blood in such patients is frequently negative, even when active lesions of a secondary type are present. This is in line with the observation noted by us [7] several years ago, that seronegative or "prematurely Wassermann negative" secondary syphilis may indicate a lack of resistance to syphilitic infection on the part of the patient, as evidenced by a heightened incidence of secondary relapses.

From this discussion it seems probable that in Germany the reaction to treatment with arsphenamine in early syphilis is less favorable now than in the years before the World War, in the sense that secondary relapses are more frequent, the Wassermann reaction of the blood is not so rapidly reduced to negative, and an increasing number of patients are appearing whose lesions actually fail to heal under arsphenamine. The explanation for these phenomena is not yet clear, but it seems probable from a study of the literature that it rests not with the drug or with a specially resistant strain of organism but, rather, with the reaction of the individual patient to infection.

Our own material at the Johns Hopkins Hospital offers an unparalleled opportunity to study carefully the response of patients with early syphilis to treatment. From 1914 on, our scheme of treatment has been essentially the same, consisting of courses of arsphenamine alternating with courses of mercury by inunction (or, since 1925, with courses of intramuscular injections of an insoluble bismuth salt). The first course of arsphenamine, until 1925, consisted of from 6 to 8 injections at a dosage level of 0.3 Gm. for women and 0.4 Gm. for men. Since 1925, the first three injections of this course have been at the level of 0.1 Gm. per twenty-five pounds of body weight, with a maximum of 0.7 Gm., subsequent injections being at the original levels of 0.3 and 0.4 Gm. Treatment is continuous, no rest periods being allowed.

A Wassermann test of the blood is done at the time of each intravenous injection (weekly), and the same technic has been followed since 1914. In chart 1 is shown the composite [8] Wassermann response (water bath incubation) to the first course of treatment in 723 patients with early syphilis treated from 1914 to 1923, and in 393 patients treated from 1925 to 1927. These curves afford a satisfactory index of the

7. Moore, J. E., and Kemp, J. E.: The Treatment of Early Syphilis: III. The Wassermann Reaction in Treated Early Syphilis, Bull. Johns Hopkins Hosp. **39**:36, 1926.

8. The method of arriving at composite Wassermann curves is described in the article referred to in footnote 6.

immediate response to treatment. Since, in the latter period, the arsphenamine dosage was higher (for the first three injections), and since the Wassermann curve did not come down toward negative as quickly as in the period from 1914 to 1923, especially following the first three injections, it might easily be concluded that early syphilis is more resistant to treatment now than then. That this is more probably due to statistical variation which is not significant is illustrated by chart 2, in which the years from 1914 to 1927 are divided into four periods. Two large and uncontrollable variables enter into any computation of this nature: the individual differences in the reaction of human beings

Chart 1.—The composite Wassermann response to one course of arsphenamine in 393 cases of early syphilis in the period from 1925 to 1927 (line and circle) as compared with that in 723 cases in the period from 1914 to 1923 (line and dot).

to syphilitic infection (noteworthy examples of which are afforded by race, sex and the influence of pregnancy) and the slight but constant variation in a biologic test such as the Wassermann reaction. Study of chart 2 must lead one to conclude that, from the standpoint of Wassermann response to the first course of arsphenamine, there is no definite evidence of increasing resistance to treatment in our material.

Chart 3 shows the Wassermann response throughout four courses of treatment of forty-three patients with early syphilis given the larger dosage of arsphenamine plus interim bismuth (1925-1927), as compared with the response of fifty treated by the original dosage of arsphenamine plus interim mercury (1914-1923). There is obviously no striking

difference, thus illustrating the fact that the Wassermann reaction in early syphilis in our clinic at present is exactly what it was several years ago. Incidentally, these curves show no material advantage for larger arsphenamine dosage and interim bismuth over our original scheme of treatment. The relative merits of bismuth versus mercury in interim treatment constitute, however, a problem that depends on factors other than this one, and cannot be discussed here.

True arsphenamine-resistant syphilis is, of course, familiar to us, as it must be in any large syphilis clinic. While no definite figures are available, the total number of cases that we have seen is small (perhaps

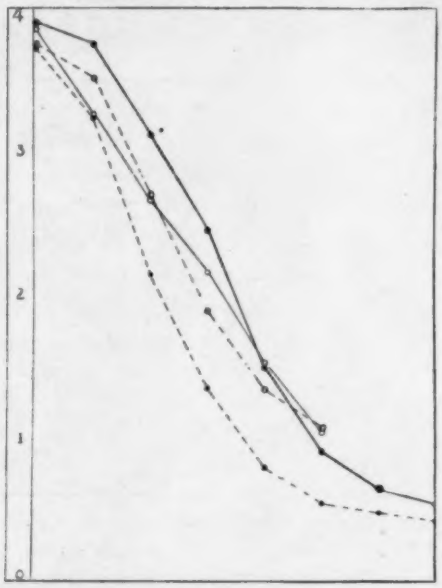

Chart 2.—The composite Wassermann response to the first course of arsphenamine in early syphilis in the years from 1914 to 1927, divided into four periods. The period from 1914 to 1917 (207 cases) is represented by a solid line and circle; the period from 1918 to 1920 (349 cases), by a broken line and circle; the period from 1921 to 1923 (170 cases), by a broken line and dot, and the period from 1925 to 1927 (393 cases) by a solid line and dot.

a dozen) and they are well scattered over the whole period of existence of our clinic. There has been no increase in recent years.

Relapses of a secondary type (cutaneous or mucosal relapses, neurorecurrences) have not been more frequent in our clinic in recent years (1925-1927) than in the years from 1914 to 1923. In each series, only those patients observed for at least one year after treatment, no matter in what amount, are regarded as free from such recurrences. On this basis, 28 per cent of 402 patients treated during the period from 1914

to 1923, and 21 per cent of 76 treated during the period from 1925 to 1927 showed such relapses.

The incidence of seronegative secondary syphilis (that is, with a completely negative Wassermann reaction or one showing less than 100 per cent fixation by water bath technic with a plain alcoholic antigen) or of "prematurely negative" Wassermann reactions (that is, the reaction becomes and remains negative after one or two injections of arsphenamine) is of interest in the two periods. In the period from 1914 to 1923, 8.2 per cent of the patients had seronegative secondary syphilis, and in 15.2 per cent the Wassermann reaction became prematurely negative during treatment. Comparable figures for the period from 1925 to 1927 are 2.3 and 9.6 per cent. If it is true that this type

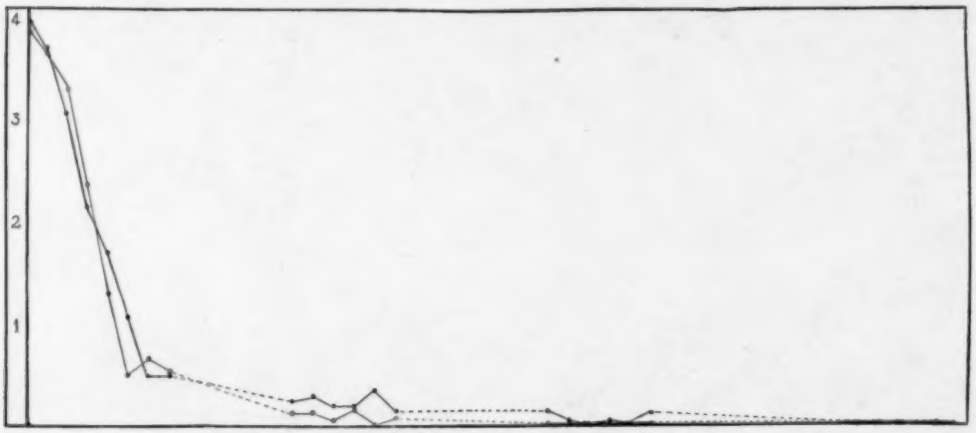

Chart 3.—The composite Wassermann response throughout four courses of treatment of a series of forty-three patients given large initial dosage of arsphenamine and bismuth as interim treatment between courses of arsphenamine (line and circle) and of another series of fifty patients treated with small initial doses of arsphenamine and mercury as interim treatment (line and dot).

of Wassermann reaction means lessened resistance to infection on the part of the patient, then there is certainly no evidence that this type of person is increasing in our material.

SUMMARY

1. A survey of the literature indicates that in Germany, and perhaps elsewhere in Europe, early syphilis reacts to treatment with arsphenamine less favorably now than in the years before the World War, in the sense that secondary relapses are more frequent, the Wassermann reaction of the blood is not so rapidly reduced to negative, and an increasing number of patients whose lesions fail to heal under treatment with arsphenamine are appearing.

2. It seems probable that this situation is due less to any decrease in the therapeutic activity of arsphenamine or to the development of so-called arsphenamine-fast strains of spirochetes than to an alteration in the reaction of the individual patient to treatment.

3. Composite Wassermann curves of 1,116 patients with early syphilis, studied from 1914 to 1927 in the syphilis clinic of the Johns Hopkins Hospital, show that there is no significant difference in serologic results in recent as compared with earlier years.

4. Relapses of a secondary type, in our material, were no more frequent in the period from 1925 to 1927 than in that from 1914 to 1923.

5. True arsphenamine-resistant syphilis is not increasing in this locality.

6. Patients with lessened resistance to syphilitic infection, as measured by the standards of "seronegative early secondary syphilis" or "premature Wassermann negativity," are no more frequent in our material now than in earlier years.

7. There is no evidence to indicate that, in this part of the United States, arsphenamine is less efficacious than formerly in the treatment of syphilis; or that arsphenamine-resistant syphilis is increasing.

8. Certain general aspects of the phenomenon of arsphenamine resistance are discussed.

DERMATITIS FROM PICRIC ACID SOLUTION AND BUTESIN PICRATE OINTMENT*

N. RILEY JACKSON, M.D.
AUSTIN, TEXAS

As I have seen no reference in the literature to picric acid as a cause of dermatitis, and in view of the fact that picric acid is used rather extensively in solution in the preparation of the field of operation and in the treatment for burns, and also in the form of an ointment (butesin picrate) as an analgesic, soothing agent, I shall report five cases of dermatitis which have come under my observation during the last few months, and which I have attributed to the irritant quality of picric acid. I do not think that this condition is found often, because it is manifested only in susceptible persons.[1]

REPORT OF CASES

CASE 1.—A student nurse, aged 20, was referred by the hospital superintendent on Nov. 17, 1928. She complained of a slightly stinging eruption scattered over the arms, body and neck, which, she said, had disappeared and recurred at intervals for the past year. The lesions were erythematous, slightly papular, more or less circular, varying in size from that of a pea to that of a dime, and were somewhat suggestive of urticarial lesions. She was asked what she considered the cause of the dermatitis, and replied that, in September, 1927, she sustained a burn on the flexor surface of the right forearm to which picric acid solution was applied for treatment. This was followed in a short time by a burning, vesicular eruption of the skin around the burn, and, in a day or so, by the same kind of eruption that she had when she came to see me. Picric acid was not reapplied and the trouble disappeared in a few days; but when she was on duty in the operating room, she noticed that every time she handled picric acid, a vesicular eruption soon developed at the site of contact with the acid solution. In a day or so similar lesions were scattered over the body. She was relieved from operating room duty for a few weeks and became well. She then consented to let me apply the same picric acid solution to an area the size of a dollar on the flexor surface of the forearm. In a few minutes an erythematous, vesicular, itching and burning dermatitis began to develop at the site of application, and in a few hours an apparently secondary, erythematous urticarial-like eruption appeared over the arms, chest and abdomen. The condition on the arm was treated with wet dressings of normal aluminum acetate solution and later with calomine lotion with menthol and phenol and cleared up in two or three days, and the secondary eruption over the body disappeared in a few days under treatment with the same lotion. I considered this scattered urticarial-like eruption the result of absorption of the picric acid solution and probably the same condition as a dermatitis medicamentosa.

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* Submitted for publication, July 22, 1929.

1. Since beginning this paper, Dr. William Allen Pusey and Dr. Herbert Rattner of Chicago have reported two cases of the same trouble in the Arch. Dermat. & Syph. **19**:917 (June) 1929.

CASE 2.—A student of the Texas State University, aged 38, single, received a rather extensive second degree burn of the left shoulder and arm down to the elbow on Feb. 6, 1929. I saw her in consultation with one of the university physicians on February 15. The burn had been dressed daily with picric acid solution and either butesin picrate ointment or unguentin. A few days after contracting the burn, she developed a marked eruption, covering almost all of the body, of the type described in case 1 as the secondary eruption. The use of picric acid was discontinued, and the wounds were dressed daily with 1 per cent surgical solution of chlorinated soda for cleansing, and parresine was applied. The eruption disappeared in a few days, and the wounds healed rapidly.

CASE 3.—A man, a university student, aged 22, was seen in consultation at Seton Infirmary. He had sustained a bruised elbow while playing baseball. The abrasion was painted with picric acid solution a day or two before I saw him. When I first saw him the elbow presented the typical appearance of a dermatitis venenata, showing an erythematous, edematous, vesicular and weeping eruption which was burning and painful and somewhat stiff. Under applications of wet dressings of normal aluminum acetate solution 1:10, the acute stage disappeared in forty-eight hours; then calomine lotion was applied for three or four days and complete healing took place. No secondary eruption appeared on other localities of the skin. The affected area was small and not deep, and practically no absorption took place.

CASE 4.—A man, a bank clerk, aged 38, referred from another town, gave a history of the appearance of a small itching area on the upper part of the arm ten days before I saw him. The lesion persisted and spread somewhat. A physician was consulted, who applied picric acid solution three times a day for two days on almost the entire length of the upper part of the arm; an erythematous, vesicular, weeping eruption appeared in due time, which also cleared up promptly under the aforementioned line of treatment.

CASE 5.—A train dispatcher, aged 40, complained of severe itching and burning of the buttocks and of a few scattered areas of the body. He presented a papular, vesicular, erythematous, weeping eruption on the buttocks and perianal area; he also showed a somewhat papular, erythematous eruption on the lower limbs, body, neck, forearms, face and margins of the scalp, all of which burned and itched a great deal. His trouble began a week previous to the time he came to my office. He gave the following history: A few weeks prior to the beginning of his present trouble, his wife was confined at one of the hospitals of the city. Following the delivery, the nurse applied butesin picrate ointment to the hemorrhoids (which is frequently done in these cases), and it gave her great relief. When she left the hospital, the nurse gave her some of this ointment to take with her for further use on the hemorrhoids. The husband had an attack of hemorrhoids, and his wife had him use some of the ointment. His hemorrhoidal condition was not relieved, and he applied more ointment; the dermatitis developed promptly in this and the surrounding area, and he applied more of the ointment and over a larger and larger area; the result over the entire buttocks was as described, and in two or three days the secondary eruption occurred. The secondary eruption in this case was somewhat similar to dermatophytids, and was probably the result of absorption of the picric acid at the original site of the trouble. The patient was sent to the hospital, and the condition cleared up in a few days under the same treatment given in the other cases.

PURPURA ANNULARIS TELANGIECTODES

CASE REPORT WITH MICROSCOPIC EXAMINATION *

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SAN FRANCISCO

In a paper that appeared in 1896, Majocchi [1] described an unusual disease of the skin characterized by the presence of minute telangiectatic puncta which entered into the formation of various sized annular lesions that disappeared in the course of time with or without atrophy.

Two additional cases formed the basis of a monograph appearing in 1904.[2] In 1905, Brandweiner [3] reported three cases; this was followed by reports of Kren [4] (1906), Arndt [5] (1907), Balzer and Galup [6] and Ferrari [7] (1908), Vignolo-Lutati [8] (1909), Sachs [9] and Brandweiner [10] (1910), Verrotti,[11] Radaeli [12] and Ossola [13] (1911), Brandweiner,[14] Lindenheim,[15] Lier,[16] Vignolo-Lutati,[17] Balina [18] and Majocchi [19] (1912), Nobl,[20] Ambrosoli [21] and Pasini [22] (1913) Truffi [23] (1914), MacKee,[24] Copelli [25] and Angelini [26] (1915) Copelli [27] (1918), Martino [28] and Semon [29] (1920), Majocchi [30] (1923) Scomarowski [31] (1925), Balzer [32] (1926), Majocchi [33] (1928) and others.

REPORT OF CASE

History.—Mrs. S. C., a saleswoman in the perfume department of a local store, aged 34, was born in Pennsylvania of Scotch-Irish parents. Both parents were dead. The mother was supposed to have had generalized neuritis previous to her death, but nothing could be learned concerning the demise of the father. One married sister lived in New York and has always been in the best of health. There was no history of tuberculosis or other chronic disease in the family.

The patient had diphtheria at the age of 10 and later varicella, whooping cough and three attacks of measles. During the attack of diphtheria antitoxin was given, and recovery was followed by premature menstruation. Ever since the menses began, the patient had suffered from nausea and an intense frontal headache just before or during the period. Although twice married, she had never been pregnant.

Four or five months before the eruption began, there was a gaseous distention of the stomach which occurred regularly one half hour after eating. The last menstrual period before the skin eruption appeared was ten days late; the second month after the appearance of the eruption, it was late again; with these exceptions, it had always been regular.

The eruption first appeared on the legs just above the knees early in March, 1928. During a visit to Arizona in May, where extremely warm weather was encountered, other lesions began to appear rapidly so that about two weeks later the eruption was profuse.

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* Submitted for publication, July 16, 1929.

* From the Dermatology Clinic of Dr. H. E. Alderson, Stanford University Medical School.

According to the patient's statement, each lesion began as a pinpoint spot, increasing in size by peripheral extension and clearing up in the center. It was her opinion that the average sized annular lesion developed from a minute spot in about ten days. When the patient was first seen on June 4, 1928, the eruption was profuse, and all stages except the atrophic were present.

Examination.—The patient was of normal intelligence and was apparently free from neurotic tendencies. She was 5½ feet (153.6 cm.) tall and weighed 126 pounds (57.2 Kg.). No stigmas of hereditary syphilis or tuberculosis were observed. The skin was somewhat lax for a person of this age, and its texture resembled that found in later life. Minute, superficial varices were especially prominent in those areas in which the eruption predominated. Just how much

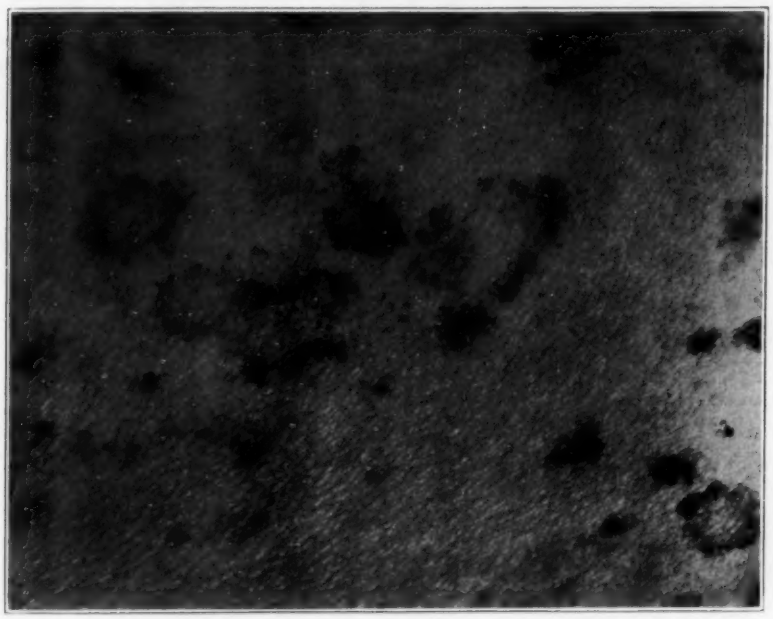

Fig. 1.—Photograph represents an area from the inner aspect of the right thigh, normal size. A biopsy specimen from this site included an early and late lesion and furnished the material studied and presented in this paper.

the fact that the patient's occupation required her to stand all day had to do with this is problematic.

Description of Eruption.—The eruption was located on the various surfaces of the legs and thighs but was limited chiefly to the thighs and appeared in greatest profusion on their inner aspect. The mucous membranes and other parts of the body were not involved.

The earliest lesions were punctate and consisted of discrete, bright red spots varying in size from that of a pinpoint to that of a pinhead, and no evidence could be seen suggesting a relationship to the hair follicles. They were not elevated and failed to disappear completely when examined by the diascope. The minute telangiectatic puncta or dots which formed the so-called red spots increased in number so that a dozen or more such points might be seen in a brownish, pigmented macule-like lesion. It was through the peripheral formation of new

telangiectatic puncta and the disappearance of those located centrally that the next stage developed. This was characterized by annular lesions which showed considerable variation in size, some being as small as 3 mm. in diameter, while others were fully 3 cm. The centers showed variation in color from yellowish to brownish in the early lesions to pale brown or normal color of the skin in the late lesions. The borders appeared telangiectatic and seemed to be made up of dilated or thrombotic capillary loops varying in color from dark to bright red, depending

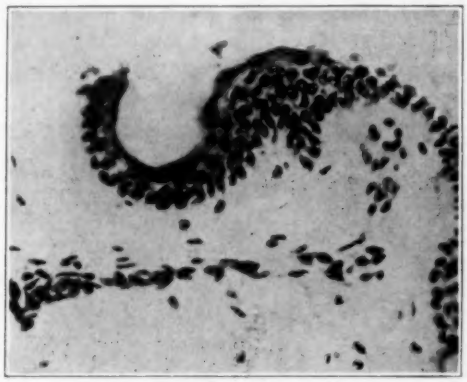

Fig. 2.—A slight dilatation of a subpapillary vessel and its papillary branch.

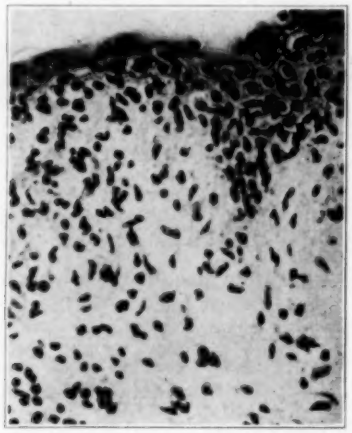

Fig. 3.—Edema with extensive cellular infiltration.

somewhat on their age. Rarely could furfuraceous scaling be detected. The terminal lesions were represented in some cases by pale, brownish, pigmented spots which seemed to disappear in the course of time without clinical evidence of atrophy.

Laboratory Examinations.—Two Wassermann reactions of the blood and a Pirquet test were negative. The blood count showed: leukocytes, 7,200; erythrocytes, 3,990,000 and the platelets, 222,000. Coagulation time was five minutes. There was a slight variation in the size of the erythrocytes. The hemoglobin was

70 per cent and the color index, 0.89. The differential count was: neutrophils, 45 per cent; basophils, 1 per cent; lymphocytes, 48 per cent and transitionals, 6 per cent.

The urinalysis showed: specific gravity, 1.022; reaction, acid; no albumin, sugar and acetone. There were no crystalline or amorphous deposits. Many leukocytes and a few cocci were found but practically no erythrocytes.

Biopsy.—A large section of skin containing the early and late stages of the disease was removed from the inner aspect of the right thigh for microscopic

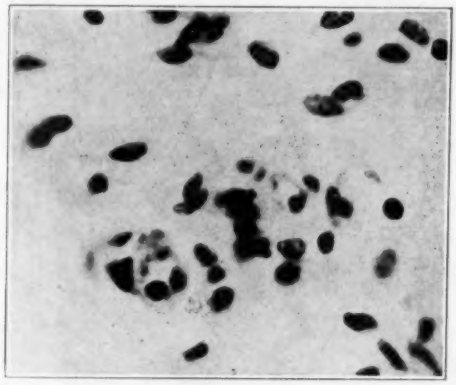

Fig. 4.—Lymphocytes, endothelial and spindle cells surrounding a vessel showing endothelial hypoplasia.

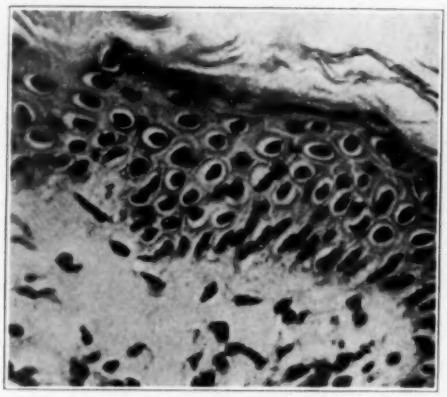

Fig. 5.—Perinuclear vacuolization of the epidermis.

study. The tissue was divided, carefully labeled and fixed in alcohol, embedded in paraffin, cut serially and stained. Serial sections stained by the following methods were studied: hematoxylin-eosin, orcein, polychrome methylene blue, Pappenheim's, Ziehl-Neelsen's, Gram iodine and Perl's Berlin blue reaction for hemosiderin.

Microscopic Changes.—The earliest change was a dilatation of the vessels (fig. 2) forming the subpapillary plexus and its terminal papillary branches. This was soon followed by a perivascular cellular infiltration composed of lymphocytes, endothelial and occasionally plasma cells. This vascular dilatation was accom-

panied by edema (fig. 3) with a resulting wide spacing of the interfascicular bundles of collagen, most marked in the papillary layer but also present in the deeper portions of the corium.

As this condition persisted an endarteritis (fig. 4) which was often obliterative developed, and then certain changes took place in the epidermis. The epidermal intercellular space became widened, and certain areas showed perinuclear vacuolization (fig. 5). Because of interference with the nutrition of the cells, various stages of epidermal necrosis were to be seen and because of this interference with the normal evolution of the epithelial cells, the stratum corneum became somewhat thickened and exfoliation might have occurred. Highman [34] mentioned the fact

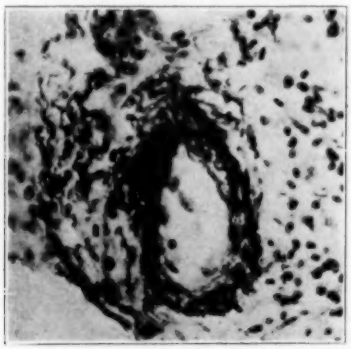

Fig. 6.—Small arteriole showing endarteritis with increased elastic fiber content.

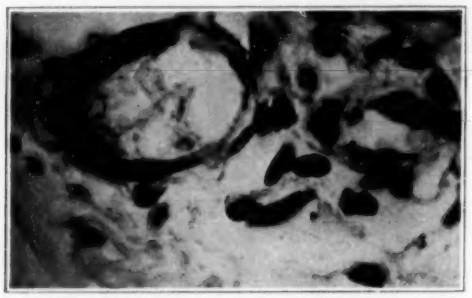

Fig. 7.—Beginning lateral thrombosis in an arteriole.

that a fine desquamation may occur, and in a case of Alderson's,[35] this feature was quite marked.

As a result of these changes, there was a gradual flattening and disappearance of the epithelial interpapillary processes, and the upper corium showed an increased fibro-connective tissue content.

Before the completion of the foregoing atrophic changes, certain vascular changes took place. The tunica intima and the tunica media including the elastic fibers (fig. 6) became swollen, and as a result of their increased thickness, the lumen of the vessel was reduced. This frequently led to thrombosis (figs. 7 and 8) and obliteration and even hyaline degeneration of the arterial wall. In purpura annularis telangiectodes, the process of vascular obliteration is slow and

continuous and never leads to a destructive necrosis because the nutrition is sufficiently maintained by the contiguous vessels. With this impairment of the circulation, the atrophic changes continue and some of the coil glands show a mucinous degeneration. In several of the sections, dilated sweat glands were seen but when the late lesions were found, that is, in the center of the annular lesions and in the fading pigmented spots, neither ducts nor glands could be seen. At the periphery of the annular lesion, the capillaries were dilated (fig. 9) but not increased in number, while in the center there was an apparent decrease.

Fig. 8.—A partly thrombosed vessel containing numerous erythrocytes.

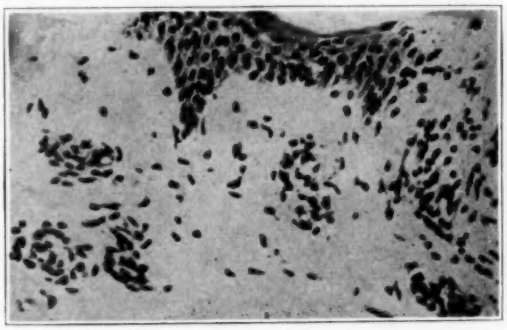

Fig. 9.—A beginning dilatation of the capillaries which are surrounded by infiltrating cells.

Hemorrhage as a result of capillary rupture or diapedesis existed to such a slight extent that it was impossible to demonstrate hemosiderin by Perl's Berlin blue reaction, although other observers have strongly emphasized its presence. Minute capillaries terminating in aneurysmal-like sacs mentioned in MacKee's excellent article were seen now and then.

The elastic fibers (fig. 10) of the dermis showed disintegration, and their disappearance or decrease was greatest where the most dense cellular infiltration and edema had occurred.

The arrectores pilorium muscles and the sebaceous glands (fig. 11) showed extensive degenerative and atrophic changes.

The nonhematogenous pigment (fig. 12) seen occurred in that part of the section in which the active changes were taking place, and while the basal epidermal cells contained most of the granules, a few were observed in the corium.

Sections stained by the Ziehl-Neelsen's method were examined by ordinary light and by Hoffmann's [36] darkfield technic. In several, acid-fast bacilli (fig. 13) were found located in the subpapillary layer of the corium. Although these corresponded in size with Koch's bacillus, because of the partial absence of the usual

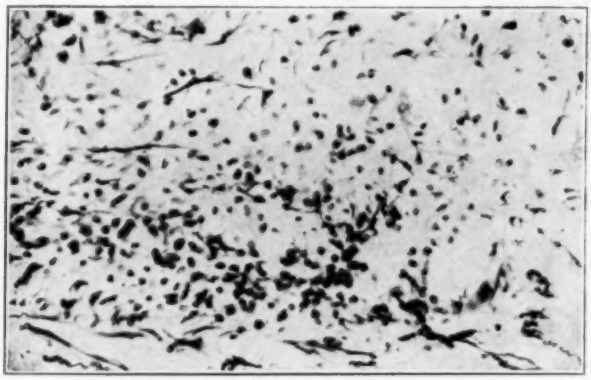

Fig. 10.—The elastic fibers are decreased in the infiltrated areas.

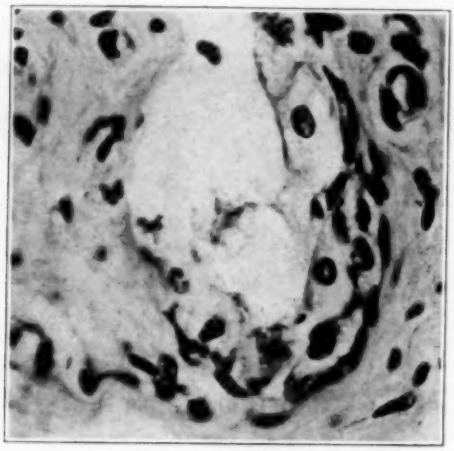

Fig. 11.—A sebaceous gland showing degenerative changes.

accompaning pathologic change seen in tuberculous tissue I do not feel justified in calling them other than acid-fast bacilli.

Russell Fuchsin Bodies: The occurrence of numerous Russell fuchsin bodies in the basal epidermal cells and the adjacent corium was most interesting. Although these bodies took many different stains, carbolfuchsin seemed to bring them out most clearly.

Because of the confusion in the literature regarding the so-called Russell fuchsin bodies, it may be well to give the generally accepted description. These

bodies are round or oval on section, varying in size from that of an eosinophil granule to two or three times the diameter of a red blood corpuscle. They are highly refractile and sharply contoured, occurring singly or more commonly in clusters of variable sizes both intracellularly and extracellularly. They showed a strong affinity for acid stains.

The Russell fuchsin bodies were first described by Fox [37] in 1858, and in 1890 Russell [38] described them as organisms characteristic of cancer. While studying the growth of cancer in various organs, Russell first noticed round or oval bodies

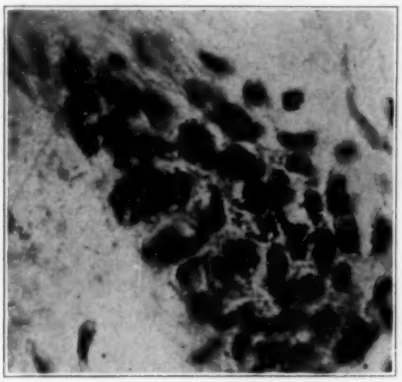

Fig. 12.—Intercellular and intracellular pigment granules in an interpapillary process.

Fig. 13.—A group of acid-fast bacilli.

which stained with fuchsin and iodine green. Having eliminated as far as possible all chances of contamination by impurities, he wondered whether they were the nuclei of tissue cells in exaggerated, formative and reproductive activity or whether they were simple globes of some form of degeneration. Because of their perfect roundness and homogenous, hyaline-like structure, it seemed impossible that they could be nuclei.

Brown [39] (1910) said that after fifty years, little real progress has been made in the solution of their true pathologic significance. Such widely divergent views

are maintained by leading workers that it is practically impossible for one to gain any clear conception of the subject from a review of the literature.

Dubreuil and Favre[40] brought out the fact that although the Russell fuchsin bodies are regarded as products of degeneration yet their presence is not always accompanied by signs of degeneration, and nothing is known concerning the genesis of these bodies except that they appear in chronic inflammatory processes.

Among the many theories advanced to explain their origin and nature, two have become more generally accepted: (1) their origin from cell granules, especially those of plasma cells, mast cells and leukocytes;[41] (2) their origin from red blood corpuscles.[42]

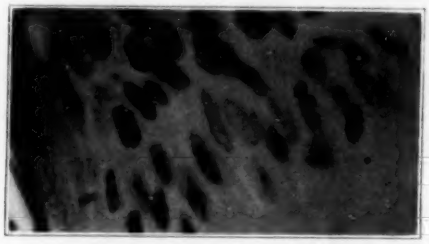

Fig. 14.—Intracellular formation of the Russell fuchsin bodies. Two of these bodies may be seen in the center of the photograph.

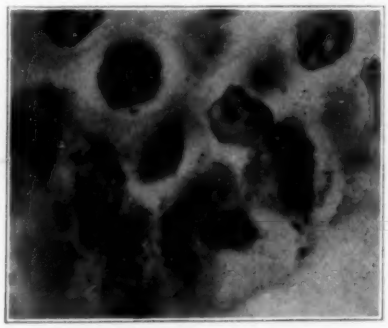

Fig. 15.—One adult Russell body occurring intracellularly and three others which are extracellular. The presence of pigment granules in their immediate vicinity should be noted.

Brown's method of staining is as follows: Fix tissue in solution of formaldehyde or alcohol. Stain first in Delafield's hematoxylin, then for five minutes in a 2 per cent aqueous solution of acid fuchsin. Transfer to compound solution of iodine (Lugol's solution) for from five to ten seconds; wash quickly in water or 95 per cent alcohol to remove the iodine. Stain from five to twenty seconds in a saturated aqueous solution of orange G. Dehydrate with alcohol, clear with xylene and mount in balsam. By this method fuchsin bodies, red corpuscles (fixed in solution of formaldehyde) and eosinophil granules stain a brilliant cherry red; nuclei stain blue, and cell protoplasm buff to orange.

Brown said that there is no single pathologic condition or class of conditions in which fuchsin bodies occur exclusively, although they are frequently found in

tumors, rhinoscleroma, granulation tissue, plasma cells and connective tissue cells. While their occurrence within cells other than plasma cells is comparatively rare, there is no question but that typical fuchsin bodies can be found in other types of cells. It has been pointed out that the areas in which they occur most numerously are those in which granulation tissue, plasma cells and newly formed capillaries occur.

Von Mueller emphasized the fact that the genesis of these Russell bodies varies and stated that they may arise not only from plasma cells but from other cells.

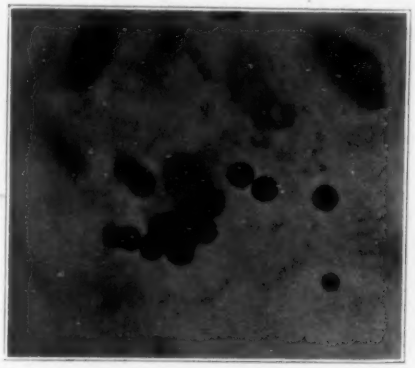

Fig. 16.—A large group of the Russell fuchsin bodies.

Fig. 17.—Suggestive extracellular reproduction of the Russell bodies by direct division.

Because of the variation in appearance, one is hardly justified in labeling any one type as a true Russell body and another as a pseudo-Russell body, as they may both possess many characteristics similar to those originally described by Russell. It is interesting to note that von Mueller believed that the Russell bodies of sarcoma are products of a hyaline cell degeneration, and said that the process of their development is very likely identical with that seen in the kidney cells and described in the literature as colloid degeneration of the kidney's epithelial cells.

In the case under discussion, the Russell bodies first appeared in the epidermis (fig. 14), either in the basement epithelial cells or in those adjoining and closely resembled the cell granules. Pigment seems to play a part in their production either directly or indirectly, and the photomicrographs of these bodies show them occurring in areas rich in pigment. An epithelial cell is capable of producing at the same time from one to two (rarely more) of these fuchsin bodies (fig. 15), which even when occurring intracellularly have a clear space surrounding them, and these bodies after maturing are apparently able to leave the mother cell without visibly damaging it. Groups containing more than two or three of these Russell bodies were not found in the epidermal layer, but in the corium as many as fourteen (fig. 16) were seen at one time in close proximity to one another.

Fig. 18.—A perfectly rounded fuchsin body. The surrounding clear space should be noted.

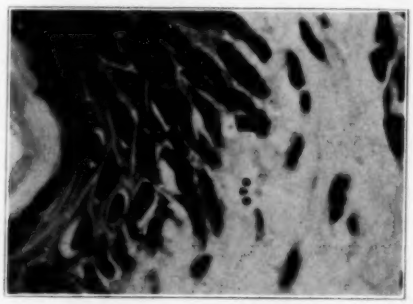

Fig. 19.—Group of three bodies in the upper corium. The clear space surrounding them should be noted.

There is reason to believe that these bodies increase in number by direct division as soon as they become extracellular (fig. 17), and this peculiarity has led many observers to regard them as of fungus origin. Under high magnification, the Russell body appears to have a clear space (figs. 18 and 19) surrounding it which in turn appears to be bounded by a capsule. Russell thought that this clear space contains a structureless transparent substance. The photomicrographs convey the impression that there is a definite clear space around these bodies but fail to support the limiting membrane or capsule theory.

If the Russell fuchsin body is the result of a degenerative process, then one would expect to find other evidence of degeneration where they are formed, but this is frequently far from the truth. While there is not always a relationship

between fuchsin bodies and degenerations, their distribution in various tissues shows a definite relation to blood vessels, red blood corpuscles and to pigment derived from them and to other pigment of nonhematogenous origin.

Brown said that the presence of some type of hematogenous pigment is practically constant; although a relationship between such pigment and fuchsin bodies is not always traceable, there are cases in which it is so clearly associated that its presence cannot be disregarded.

The occurrence of fuchsin bodies in cells other than plasma cells, their occurrence in and near blood vessels, their association with pigment, especially the hematogenous type, and the similarity in the staining reactions of fuchsin bodies and red blood corpuscles are points deserving special emphasis. However, my observations led me to believe that if pigment is a necessary requisite in the production of fuchsin bodies, the nonhematogenous form present in this case was equally productive.

Fig. 20.—The photomicrograph represents the center of one of the annular lesions and shows a disappearance of the papillae and a thinning of the epidermis.

COMMENT

The primary changes in purpura annularis telangiectodes occur in the corium, while those of the epidermis are secondary and representative of the pathologic changes seen in many of the more common dermatoses.

The beginning of the annular lesion as a minute papule and its increase in size by peripheral extension seem to be confirmed by the fact that the oldest pathologic changes were found in the center of a fully developed lesion.

It becomes apparent as one delves into the pathology that local causes can be excluded, and that the toxic theory advocated by Majocchi and many other observers deserves attention.

The earliest pathologic change, capillary dilatation, suggests a toxic origin. In the corium, the vascular dilatation, the endarteritis, the edema and lymphocytic infiltration are in accordance with the toxic

theory of the disease. Contrary to the observations of other workers, the capillaries in this case, although dilated, are not increased in number.

Majocchi's belief that these toxins are tuberculous in origin has never been fully substantiated, although in my case acid-fast bacilli were found. From a careful study of the sections, it is impossible to believe that hemorrhage is characteristic of this disease.

Unlike the case of Scholtz,[43] the prussian blue stain in my case was repeatedly negative, and this result agreed with the microscopic observations. Although atrophy could not be demonstrated clinically, it was apparent microscopically, as the photomicrographs show (fig. 20).

Just what part, if any, the Russell fuchsin bodies play in the disease is problematic. All the necessary requisites for their formation exist in this disease, namely, plasma cells, pigment and evidence of degeneration.

Outside of high lymphocyte count of 48 per cent, which is possibly suggestive of tuberculosis, the laboratory reports show little variation from the normal.

The history of the gastro-intestinal disturbances prior to the appearance of the eruption again brings us to the toxic but not necessarily to the tuberculous theory.

The relationship between the delayed menstrual period before the onset of the eruption, the one occurring during the second month after the onset and the disease in question is a matter of conjecture, but it seems highly probable that there is some connection.

There appears to be no doubt that Majocchi's disease is a definite clinical entity. Its toxic origin is in accordance with the pathologic changes, but definite evidence seems wanting to prove that it is due to a tuberculous infection. In the case reported in this paper, the patient has been under close observation since June 4, 1928. Following her first visit, the lesions have been gradually undergoing evolution and disappearing (without medication) so that when last seen, one year later, all evidence of the disease had disappeared and the atrophy mentioned by some observers was not apparent.

SUMMARY

1. The microscopic changes suggest that the disease is due to a toxin.

2. Acid-fast bacilli were found, but doubt of their being Koch's bacilli exists.

3. Intradermal hemorrhage is not a constant factor in this disease.

4. Just what significance the Russell fuchsin bodies have in purpura annularis telangiectodes is problematic. Their presence in previously reported cases, as far as I know, has not been mentioned, and it seems difficult to regard them as mere products of degeneration.

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LOCALIZED SOLID EDEMA OF THE EXTREMITIES IN ASSOCIATION WITH EXOPHTHALMIC GOITER*

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The relationship between the glands of internal secretion and certain types of dermatosis that accompany dysfunction of these glands is little understood. Among the best known of the clinical pictures is myxedema, the result of hypofunctioning of the thyroid gland. The changes in the skin associated with the hyperfunctional disturbances of the thyroid gland are not so characteristic nor so pathognomonic and are less frequently recognized. In spite of the fact that myxedema is not an exceptionally rare disease, little is known of the mechanism or of the chemistry of the cutaneous deposit which produces the characteristic clinical appearance. It is my purpose in reporting this series of eight cases, which presented identical cutaneous complications, in association with hyperthyroidism of the exophthalmic goiter type, not only to call the attention of dermatologists to this entity but also to discuss the clinical and the microscopic features of these cases in their relationship to myxedema. I believe, too, that with the scanty knowledge of dermatologic endocrinology it behooves dermatologists to report cases that relate directly or indirectly to internal glandular dysfunction.

The term "localized myxedema" has been applied by several authors to a variety of degrees and types of edema that have appeared in association with dysfunction of the thyroid gland. Most of these reports refer to edema of the face, upper part of the chest and scattered areas over the trunk. Frequent references to the development of nonpitting edema of the eyelids following thyroidectomy for exophthalmic goiter are to be found in the literature. Reports have also appeared in which the localized patches of edema were situated on the lower extremities, as well as on the face and trunk, in association with disease of the thyroid gland. The cases I shall report deal only with the development of these plaques on the lower extremities; therefore, edema of the face and upper part of the chest will not be included in the discussion. However, in passing, I believe it worth comment that in the publications relative to localized myxedema of the face and chest, evidence is lacking to show that the authors excluded in the

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differential diagnoses such diseases as acute edematous scleroderma, solid edema of the face and edema secondary to recurrent lymphangitis of the face. In the description of one published report of a case of localized myxedema of the trunk, the evidence strongly favored a diagnosis of morphea. In practically all of the reports of cases of localized myxedema to which I have had access there has been questionable evidence of thyroid hypofunction.

It is Plummer's [1] belief that edema of the upper part of the trunk, the head and particularly of the eyelids which develops in patients with symptoms of hyperthyroidism is not a manifestation of myxedema. He does not recognize this type of edema as evidence of hypofunction of the thyroid gland.

In 1927, Richter [2] reported a case of localized myxedema with reference to exophthalmic goiter and reviewed the literature. The clinical features and course of events in Richter's case closely resembled those in the cases in this report. Richter believed that the edematous plaques above the ankles were patches of myxedema, the result of local hypothyroidism secondary to disturbances in other glands of internal secretion and that the dysfunction of the incretory glands was attributable to the hyperthyroidism. He called this the third stage of exophthalmic goiter. He presented a microscopic description of a section removed from one of the plaques and attached considerable significance to the presence of edema of the connective tissue with hyperplasia of the connective tissue fibers and numerous scattered starlike cells. He believed this picture to be identical with the microscopic picture of myxedema.

Following thyroidectomy in the seven cases in this series, the patients improved materially for periods of six months or more. Then signs and symptoms of hyperthyroidism again developed, associated with the development of tawny, indurated, isolated, edematous-looking plaques; the plaques did not pit on pressure and were confined to the lower third of both legs in each case. The eighth case differs from the other seven in that the edematous plaques developed six months before the symptoms of exophthalmic goiter were recognized, and eighteen months before the thyroid vessels were ligated.

DETAILED REPORT OF FOUR OUTSTANDING CASES

CASE 1.—A school teacher, aged 23, came to The Mayo Clinic in May, 1925. She gave a history of the development of symptoms of exophthalmic goiter during the autumn of 1922, at which time she had lost 55 pounds (24.9 Kg.). Three lobectomies and two ligations had been done elsewhere between January and

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May, 1923. Following these operations, her condition had definitely improved and she had regained normal weight. During the height of the hyperthyroidism, she had marked edema of both ankles. The edema had persisted for approximately six months after the operations. In June, 1924, about one year after the last operation, indurated, tawny plaques had developed on the outer side of each ankle (fig. 1). At first these had been transitory and had tended to disappear in the morning or after a rest of a few days in bed. After about six months, however, the edematous-appearing plaques had become permanent and did not diminish with rest. There had been slight pain in the plaques associated with standing for long periods, and the patient had been concerned because of cold feet.

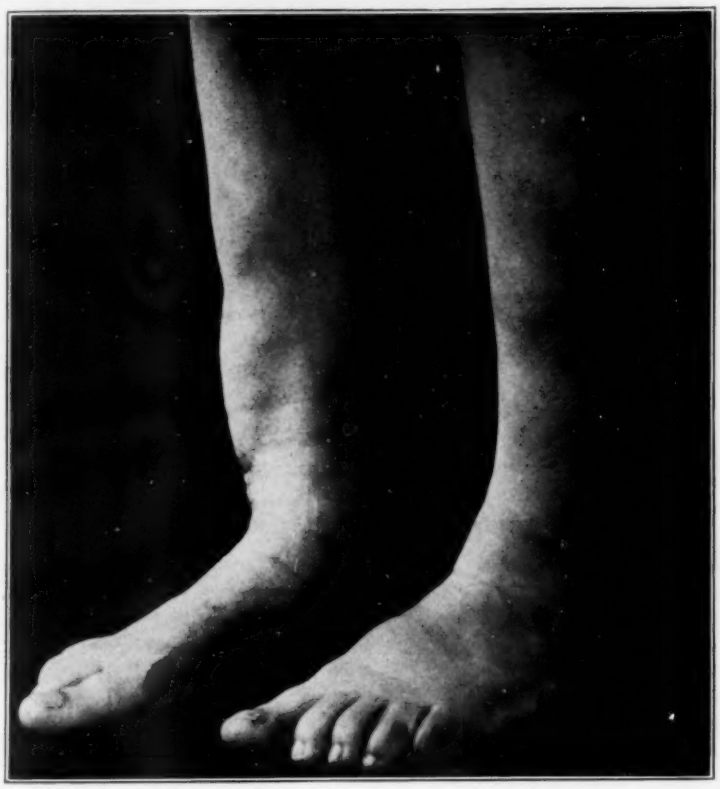

Fig. 1 (case 1).—Site of the plaques; absence of edema in the foot and leg may be noted.

On the patient's admission to the clinic, residual symptoms of hyperthyroidism of the exophthalmic type were noted. The heart sounds were normal; thrills or bruits were not elicited. The basal metabolic rates were plus 21 and plus 17 per cent (DuBois standard). The use of compound solution of iodine improved the general condition somewhat, but did not alter the plaques on the legs.

On the outer side of both lower extremities, from the external malleoli a third of the way up the legs, were tawny, nonpitting, circumscribed, slightly reddened, elevated plaques, approximately 15 cm. long and 6 cm. wide. The pigskin-like character of the lesion is shown in figure 2. There were several smaller and

softer patches on the inner side of the left ankle. The upper eyelids were slightly puffy. The use of ten drops of compound solution of iodine, twice a day for a month, improved the general condition but did not change the edematous plaques. The arms and fingers were not affected. Because the patient was greatly upset by the appearance of her ankles the lesions were excised. Culture of a piece of the excised tissue did not produce growth of bacteria.

The patient returned six months after the excision of the plaques, during which time the symptoms of hyperthyroidism had recurred and the plaquelike swellings had begun to reappear in the scars. She had been taking ten drops of

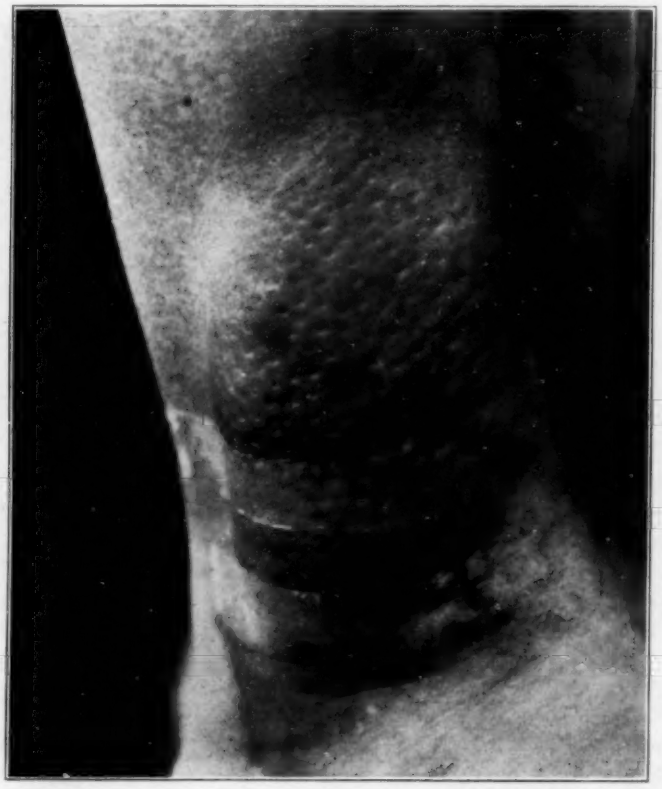

Fig. 2 (case 1).—A close view to show the pigskin-like nature of the plaques. The wound at the lower border of the plaque was made at biopsy.

compound solution of iodine twice a day, irregularly, with but slight improvement in her general condition. She returned at this time because her mother also had symptoms of exophthalmic goiter. The basal metabolic rate was plus 20 per cent and a mild degree of myocardial degeneration was noted. Because of the symptoms of recurrent exophthalmic goiter another lobectomy under local anesthesia was advised, but the patient refused to undergo the operation.

Further study of the areas from which the plaques had been excised showed that the suspected recurrences were keloids. They were materially improved by the use of radium. During the last three years there has been no further recurrence of the tawny plaques.

The microscopic examination of the excised plaques disclosed (fig. 3) slight irregularity of the rete ridges in the epidermis and in some places a mild degree of hyperkeratosis. Some of the basal cells had taken the eosin stain in their cytoplasm, and a number were pyknotic and shriveled. Throughout the prickle cell layer there were isolated cells with almost complete vacuolization of the cytoplasm, forming tiny lacunae. A few of the basal cells showed this change.

In the stratum papillare there was fairly uniform homogenization of the connective tissue without tinctorial change or evidence of mucin. The striking

Fig. 3 (case 1).—Localized areas of hyperkeratosis, homogenized connective tissue in the stratum papillare, myxomatous changes and edema in the broad band in the middle of the cutis, and elastic fibers (stained black) in the deeper cutis (×35).

changes in the section were noted in the rest of the cutis, extending deep into the subcutaneous tissue. With the hematoxylin and eosin stain the collagen fibers appeared edematous and homogeneous. They were broken into short strands and in other areas appeared frayed and splintered. Interspersed between the collagen fibers and overlying them was a substance staining blue in the hematoxylin-eosin preparation, which was amorphous; sometimes it appeared as thin lines which under the oil immersion lens appeared to be composed of fine granules. Irregular

masses of the substance might be found in other areas. With the mucicarmin stain this material stained red.

In the upper cutis there was a mild perivascular lymphocytic and plasma cell infiltrate and some proliferation of the endothelial lining of the blood vessels. Some of the fat cells in the subcutaneous tissue were outlined by this mucin, which not only lay between and over the collagen fibers but also seemed to have invaded the walls of some of the blood vessels in the cutis and to have surrounded the sweat glands in some places. The elastic tissue was decreased in amount throughout the cutis and was frayed, splintered and broken into short, curly strands. It was also decreased in amount about some of the blood vessels. The cutis was greatly increased in thickness throughout the entire section.

Case 2.—A man, aged 56, came under my care in April, 1919, at which time a diagnosis was made of exophthalmic goiter with adenomas, myocardial degeneration and auricular fibrillation. The patient had lost essentially 40 pounds (18.1 Kg.), was nervous, dyspneic and had cardiac palpitation and tachycardia. A rather coarse tremor with edema of both extremities and prominence of the eyes (exophthalmos) were also noted. He complained of loss of strength, a dull headache and insomnia. These symptoms had been present about six months.

The right lobe and isthmus of the thyroid gland were enlarged; the left lobe was only slightly enlarged. Thrill and bruit were heard over the upper pole of the right lobe. Auscultation of the heart sounds revealed a roughened first sound with a systolic murmur transmitted to the axilla. The pulmonary second sound was greater than the aortic second sound, and the first sound at the aortic region was obliterated by a blowing murmur. Systolic murmur was heard all over the base of the heart and was transmitted upward to the vessels of the neck. There was marked edema of both extremities. The basal metabolic rate was plus 41 per cent.

On April 15, 1919, the left superior thyroid vessels were ligated. Six days later partial thyroidectomy and double resection of the gland were done. The gland was about three and a half times the normal size; the right lobe was resected, preserving a small amount of the glandular tissue along the cricoid cartilage, in the posterior portion of the gland. The isthmus was removed. Resection of the left lobe was performed, preserving glandular tissue amounting to about a normal sized lobe. The muscles were cut on the right side. Microscopic examination showed the thyroid gland to be of the hypertrophic parenchymatous type (exophthalmic goiter). The patient's convalescence was uneventful.

In October, 1919, the patient returned to the clinic because of edema of the fingers and localized swellings of the lower part of the legs of recent onset (fig. 4). Symptoms of hyperthyroidism had also recurred. He reported that he had been perfectly well for five months following thyroidectomy. With the recurrence of symptoms of hyperthyroidism, the cardiac complaint had not returned. The basal metabolic rate at this time was plus 30 per cent. Dermatologic examination disclosed a nonpitting edema-like appearance of the fingers. There were, above both ankles, indurated, slightly erythematous plaques, which were tawny but with no evidence of ulceration. The plaques were 15 cm. long and 12 cm. wide, and were raised about 3 or 4 cm. above the normal skin. Roentgenographic examinations revealed low-grade periostitis of the tibia and cystic changes in the phalanges. Because of the periostitis, a therapeutic test for syphilis was given without benefit. A satisfactory explanation for the cystic changes in the phalanges was not made. Repeated examinations for *Filaria sanguinis-hominis* gave negative results.

A diagnosis of recurrent exophthalmic goiter was made, and another resection of the left lobe of the thyroid gland was done in March, 1921. At this operation the left lobe, which was found to be two times the normal size, was resected; however, enough glandular tissue was preserved to equal one-sixth the size of a normal lobe. The pathologic report again was of hypertrophic parenchymatous thyroid tissue of the exophthalmic goiter type. The use of 10 mg. of thyroxin intravenously, for three successive days, at this time, did not alter the edematous plaques.

The patient was seen again three years later and complained of being weak, tired, with a nervous exhaustion type of complex, anxious and somewhat

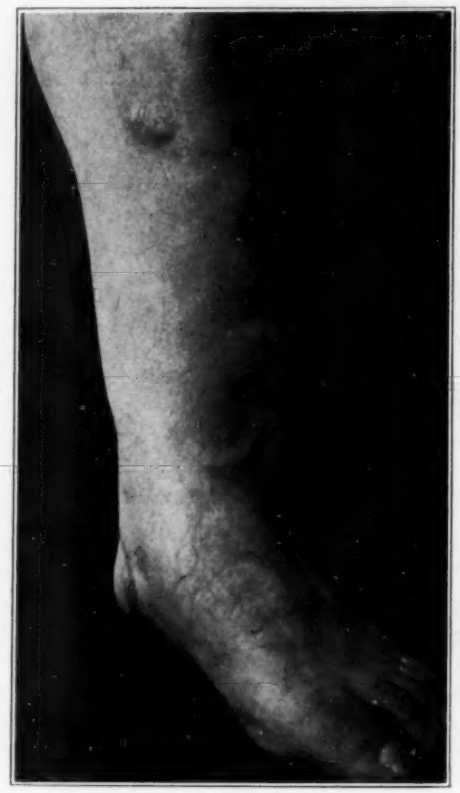

Fig. 4 (case 2).—The degree of the infiltration of the plaques is shown.

depressed. He showed a moderate amount of tremor and exophthalmos. Compound solution of iodine, 10 minims (0.6 cc.) given twice a day for three days, improved the patient's condition considerably. The basal metabolic rate at this time was plus 10 per cent. Frequent observations thereafter showed a rate varying between plus 10 and plus 20 per cent. The patient derived considerable benefit from the use of compound solution of iodine. He again came under observation in November, 1925, when the basal metabolic rate was plus 16 per cent, and there was a mass in the left lower portion of the abdomen attached to the ileum. Roentgenographic examination of the colon gave negative results. Auricular fibrillation in addition to cardiac hypertrophy and mild hypertension was

noted. The patient refused surgical intervention for the abdominal mass. In a recent communication he reported that the plaques had decreased somewhat and the swelling in the fingers and the toes had disappeared.

A microscopic study of a section removed from one of the plaques revealed more inflammatory reaction about the blood vessels than that found in the other cases (fig. 5). A few dilated lymphatics could be seen, although the lymph spaces and not the lymph vessels showed more evidence of dilatation. There were a few eosinophils present in the infiltrate; such cells were not noted in the other cases. The remainder of the section was similar to that in case 1.

Case 3.—A woman, aged 24, first came under my care in October, 1916, when she presented tuberculous cervical lymph nodes, quiescent pulmonary tuberculosis

Fig. 5 (case 2).—Perivascular infiltration in the upper half of the cutis (× 120).

and papulonecrotic tuberculids of the fingertips (folliclis) and lower part of the legs (erythema induratum). After microscopic examination the cervical lymph nodes were reported to be tuberculous. Under my direction she received a course of neoarsphenamine, for its nonsyphilitic effect on the tuberculids. This was followed by application of the roentgen rays and radium to the cervical region. There followed definite improvement in both the tuberculids and the tuberculous lymph nodes. In 1923, the patient returned to the clinic because of rectal ulceration; a microscopic section from this region was reported to be tuberculous. The erythema induratum had recurred, however; following another course of neoarsphenamine, the ulcerations of the legs healed rapidly.

In November, 1926, the patient again returned, presenting symptoms of hyperthyroidism of the type of exophthalmic goiter. The basal metabolic rate was plus 32 per cent. A microscopic section following subtotal thyroidectomy revealed that the tissue was hypertrophic parenchymatous thyroid of the type of exophthalmic goiter. The operation and convalescence were uneventful. Four months after the thyroidectomy she returned to the clinic with a recurrence of the symptoms of hyperthyroidism. The basal metabolic rate was plus 52 per cent; there had been a loss of 12 pounds (5.4 Kg.) and a diagnosis of recurrent exophthalmic goiter was made. At this time the patient presented a new form of nodule on the legs (fig. 6). She had been accustomed to the lesions of erythema induratum and to

Fig. 6 (case 3).—The infiltrated plaques which have developed in the scars that have resulted from the erythema induratum.

the ulcerations associated with them, but the tumors that were developing in the scars of the former lesions of erythema induratum she felt were different. These tumors had been noted first about one month after the thyroidectomy.

The lesions on the lower limbs differed from those in the other cases in that the nodules were smaller, more numerous and confined to the scarred areas on the lower extremities. The lesions did not pit on pressure, and the patient said that a month's rest in bed had reduced the swellings only slightly. Because of the recurrent symptoms of hyperthyroidism, a second partial thyroidectomy was done with resection of the right lobe. A piece of glandular tissue, a little less than

half the size of a normal lobe, was left on the right side. The left side appeared normal in size; enlargement of the isthmus was not noted. A pathologic report of hypertrophic parenchymatous thyroid tissue was again made.

At this time there was no evidence of the presence of active tuberculids, although characteristic scars were observed on the fingertips. The cervical lymph nodes were quiescent, and there were no clinical signs of activity in the chest where tuberculosis had been recognized previously. Supportive bandages for the extremities were recommended, and although the patient had materially improved following the second thyroidectomy there had not been, at the time of writing, any material change in the size of the plaques on the legs. There was edema of the extremities, graded 2, at the time of the second thyroidectomy, although notation of the presence of the edema was not made when the first diagnosis of exophthalmic goiter was made. A section of the skin was not obtained for microscopic study.

Case 4.—A woman, aged 30, came to the clinic in September, 1923, complaining of nervousness, dyspnea, tremor, palpitation and tachycardia, loss of strength, intermittent diarrhea, increased sweating, edema of the extremities and exophthalmos. In spite of an excessive appetite, she had lost 47 pounds (21.3 Kg.) in the previous nine months. The basal metabolic rate on admission was plus 53 per cent.

At the time of admission, on the outer, middle half of each leg were erythematous, thickened plaques which did not pit on pressure. There was evidence of residual pigmentation, and the infiltration was more pronounced in the dependent portion of the plaques. Changes of significance were not noted in the surrounding skin, and the line of demarcation was rather pronounced. For several months previous to the appearance of the plaques the patient had noticed moderate edema of the extremities, which disappeared on rest in bed. Gradually, the pitting edema of the lower part of the legs had begun to localize on the outer aspects of the calves. These localized plaques had also been transitory at first but gradually had become permanent.

This case differs from the others in that the localized edematous plaques developed before the patient's symptoms had advanced far enough to warrant a diagnosis of hyperthyroidism or before the exophthalmos had been noted. Following a course of compound solution of iodine, the basal metabolic rate dropped from plus 53 to plus 39 per cent, and following ligation of the right and left superior thyroid vessels the rate decreased to plus 29 per cent. Thyroidectomy was done six months later. The patient did not have a recurrence of the hyperthyroidism. At the time of writing, the plaques were considerably reduced in size and she believed there had been slow but steady regression in the lesions since the thyroidectomy. The microscopic picture in this case was similar to that in case 1.

COMMENT

Seven of the eight cases presenting the complication which is the subject of this paper pursued an identical clinical course. A moderately severe degree of hyperthyroidism of the exophthalmic type, for which thyroidectomy was done, was followed by a varying period of decided improvement. This, in turn, was followed by recurrence of the symptoms of hyperthyroidism. The degree of hyperthyroidism in each case was severe and was associated with auricular fibrillation and edema of the lower extremities during the first attack of hyperthyroidism or

following the thyroidectomy. Case 4 differed from the rule in that the localized edematous plaques developed simultaneously with the onset of the symptoms of hyperthyroidism.

The patients who were under my care during the second attack of hyperthyroidism were benefited by compound solution of iodine, but it did not influence the localized swellings. In one case the use of thyroxin also failed to decrease the plaques around the ankles. A striking feature in each case was the slight amount of edema in the feet and uninvolved parts of the ankle after the development of the plaques. Also, features common to all were the history of a transient pitting edema of the extremities, which was relieved by rest in bed, and the fact that the localized swellings were at first also transient and decreased following rest in bed. After a period of several months, however, the plaques became permanent. In the three cases in which a second thyroidectomy was performed without recurrence of the symptoms of hyperthyroidism, the edematous plaques remained unchanged.

The significant laboratory data were secured from studies of the basal metabolic rates.

STUDIES OF METABOLISM

Estimates of the basal metabolic rates were made in all cases. At the time of acute hyperthyroidism, the rates in three of the cases varied between plus 41 and plus 32 per cent (DuBois standard). At the time when the patients returned with the localized plaques, the rates varied between plus 30 and plus 51 per cent.

Although the ingestion of compound solution of iodine decreased the metabolic rates and the acuity of the symptoms of hyperthyroidism, it did not influence the plaques on the legs.

Boothby [3] showed that the edema of myxedema is probably an albuminous colloid fluid with a nitrogen-water ratio higher than the average 1.1 per cent from human blood serum, identical with that of egg white which contains approximately 2 per cent of nitrogen, and definitely less than that of muscle which contains more than 3 per cent. He believed that the myxedematous patient has an excess of such albuminous fluid as compared with a normal person, and hence, that the edema of myxedema corresponds to an increase in the reserve or deposit protein. Myxedematous patients show an increase in the blood urea with a corresponding increase in the nonprotein nitrogen after the ingestion of thyroxin. The chemistry of the blood was not studied in these cases.

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3. Boothby, W. M.; Sandiford, Irene; Sandiford, Kathleen, and Slosse, Jean: The Effect of Thyroxin on the Respiratory and Nitrogenous Metabolism of Normal and Myxedematous Subjects, Tr. A. Am. Physicians **40**:195, 1925.

MICROSCOPIC OBSERVATIONS

The established facts in regard to the microscopic features of myxedema are few, chiefly because the microscopic changes in the skin of myxedematous patients are not uniform. The essential epidermal changes, which are of a secondary nature, are the flattening of the epidermis and the alterations in the staining reactions of the epithelial nuclei. In the cutis the collections of mucin, irregularly distributed and of varying degrees, are characteristic of the disease. Increase and swelling of the collagen bundles are also characteristic (fig. 7). The

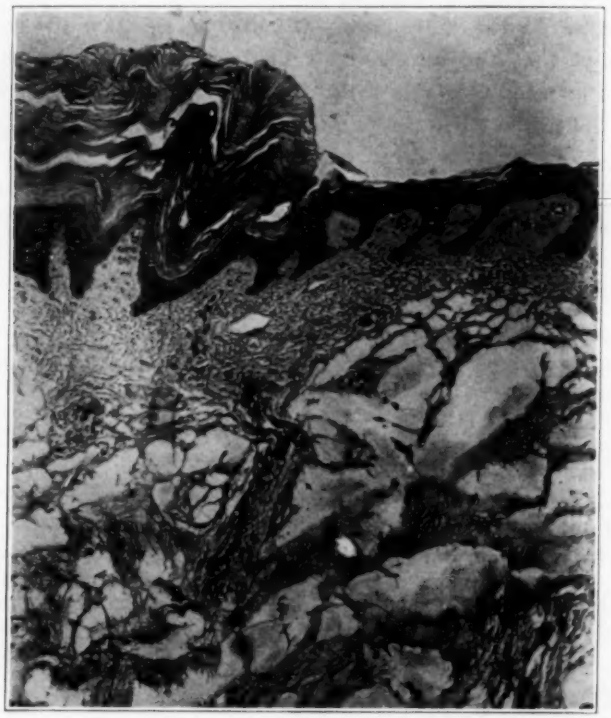

Fig. 7.—Localized hyperkeratosis, homogenized connective tissue in the stratum papillare and the myxomatous tissue beneath (hematoxylin and eosin; × 75).

changes in the elastic fibers are similar to those seen in senile degeneration of the skin. The variety of microscopic pictures that have been described may be attributed to the fact that the degree of myxedema and the stage of the disease during which the biopsy is made alter the microscopic picture. In addition is the fact that the degree of the cutaneous change does not always parallel the degree of the hypothyroidism.

The various forms of lymphedema and elephantiasis, as well as myxedema, were considered in the differential diagnosis. The published microscopic descriptions of lymphedema agree as to the essential fea-

tures. The subcutaneous tissue is the seat of the salient changes. Hypertrophy of the connective tissue, with enlargement of the blood vessels, lymph vessels and lymph spaces, and a varied degree of inflammatory reaction in and around the blood vessels, comprise the common microscopic features.

In the group of cases included in this report the microscopic picture was essentially one of an extreme degree of myxedema. In the epidermis, the thinning and atrophy of all layers except the stratum corneum is in proportion to the duration of the disease. The most marked atrophy of the epidermis was noted in one case in which the plaques had been present four years. The stratum corneum shows a relative and occasionally a marked absolute hyperkeratosis. The changes in the epidermis appear secondary to the degree of the myxomatous change and the duration of the disease.

In the stratum papillare, homogenization of the connective tissue is seen in varying amounts. The elastic tissue fibers are unchanged, but a few dilated capillaries and lymph spaces are seen in this region. In one case, deposits of mucin were found in the stratum papillare.

In the cutis, which is greatly thickened in all cases, diffuse and extensive deposits of mucin appear both in homogeneous masses and in fine strands. Under the oil immersion objective, the mucin seems to be composed of fine granules. The mucin may be seen surrounding short, broken strands of collagen fibers, about the sweat glands and within the walls of some of the smaller blood vessels. In one case the mucin appeared as a broad band in the upper part of the cutis and was pierced by blood vessels and ducts of sweat glands. The increased depth of the cutis can be accounted for in all of my cases by the edema and the myxomatous changes. The connective tissue in the cutis showed marked edema and homogenization of the fibers and, in certain areas, a fraying and splintering of the fibers. Scattered throughout the cutis, oval and stellate-shaped cells were seen between collagen fibers and surrounded by mucin.

The changes in the blood vessels were not alike in all the cases. A varying amount of cellular infiltration and edema of the vessel walls were seen. A few of the walls of the blood vessels contained mucin, and in one case there was obliterative endarteritis.

TREATMENT

The choice of treatment for the lesions on the extremities is dependent on the demands of the patient. Local measures, such as supportive bandages, were of no value. Rest in bed and other measures directed toward the treatment for the hyperthyroidism had no influence on the edematous plaques. In the young woman (case 1) to whom the cosmetic effect of the plaques was distressing, surgical

excision gave a fairly satisfactory result. A modified type of Kondoleon operation was done and was followed by the development of a hypertrophic scar, although the edematous plaques did not recur following excision.

In three of the cases the lesions have undergone spontaneous involution. The remnant of the plaques is lichenified, slightly pigmented and raised. Accordingly, observation offers a satisfactory form of treatment if the patient will tolerate the lesions three or four years.

SUMMARY

This report includes the records of eight patients in whom nonpitting, edematous, tawny plaques developed on the lower extremities in association with hyperthyroidism. The features common to all cases are: acute hyperthyroidism of moderately severe degree; long-standing edema of the extremities of cardiac origin and tawny plaques that were not influenced by the therapeutic measures which brought about improvement in the symptoms of hyperthyroidism. The microscopic picture is that of a marked degree of myxedema, although the clinical picture in each case at the time of the development of the plaques was one of acute hyperthyroidism. I do not offer any explanation at this time of the mechanism involved. I have avoided the expression "localized myxedema" in reference to my own work because I believe that the term myxedema is now, because of common usage, applicable to the group of constitutional symptoms which develop as a result of hypofunctioning of the thyroid gland and of which the cutaneous manifestations are merely a coincident phase. The study of this series of cases has precipitated a more intensive investigation into a combined physiochemical microscopic study of the cutaneous changes in myxedema.

DYSKERATOSIS CONGENITA WITH PIGMENTATION, DYSTROPHIA UNGUIS AND LEUKOKERATOSIS ORIS*

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AND

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CLEVELAND

At the annual meeting of the Cleveland Dermatological Society held in October, 1926, a case was presented for diagnosis in which the patient showed a peculiar scarflike, pigmented eruption of the neck, an acrocyanosis of the hands and feet with hyperhidrosis of the palms and soles, a dystrophy of the nails and a leukoplakia-like eruption of the tongue and hard palate. The history and description are given subsequently.

REPORT OF CASES

History.—The father, mother and three sisters were living and well. No one in the family had had the same condition as the patient, and he knew of no other relatives who had this trouble.

The patient, a man, aged 20, had had the ordinary diseases of childhood—measles, mumps and varicella. He had been troubled as far back as he could remember with what had been called undernourishment or malnutrition. He was in a severe automobile accident at the age of 13, which resulted in the shortening of one of his legs about an inch. He graduated from high school at the age of 18 and at the time of presentation was attending an art school.

The patient said that as far back as he could remember he had had a peculiar condition of his finger-nails and toe-nails, and a peculiar pigmentation over his neck. He never knew that the skin over his tongue was different from that of other people. He said that his feet perspired freely, that he also noticed a great deal of perspiration on his hands, and that his hair was rather dry. He occasionally had large blisters on the heels, which would sometimes be the size of a castor bean or even that of a half dollar. These bullae, the patient said, were difficult to break, and often a long time would elapse before they broke open.

Examination.—The pupils were equal and round and reacted well to light and accommodation. There was nothing abnormal showing either in the pupillary area or in the cornea. Numerous scars of old chickenpox were noted over the face.

Mucous Membrane: The entire dorsum of the anterior two thirds of the tongue showed a peculiar glazed, whitish appearance. The surface was rather

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smooth. The natural rugosities of the tongue did not show up so well. The surface was likewise rather shiny, and here and there, distributed over the area, one noted small, bright red spots where there was a break in the leukoplakia-like formation. This was not noted on the inside of the cheeks, but over the hard palate, in about the middle half, there was an area the size of a fifty cent piece, showing this glazed, rather whitish appearance. On the left side of this area there was one spot, the size of a pea, which was slightly raised and of a dense whitish color.

Skin: Over an area extending upward almost to the jaws, backward to just posterior to the ear, and downward in a straight line almost to the clavicles on each side and then converging from each side symmetrically to a point on the sternum about midway between the mammae, one found an eruption most profuse over the anterior portion of the neck, down to the level of the clavicle, but nevertheless involving all the areas described. It was of a peculiar, light, reddish-brown, or possibly grayish-red, tint. It was made of fine, pinhead-sized, smooth, somewhat whitish, raised papules, in linear rows, following the somites of the skin, and with a fine reddish base between these rows. These papules in their

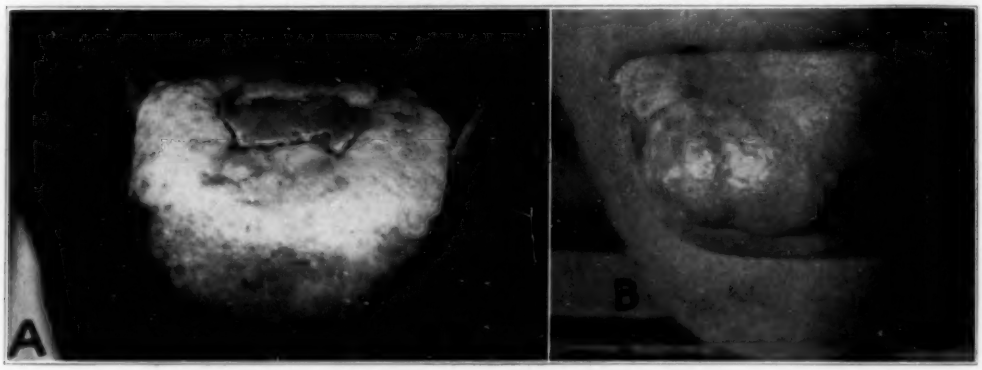

Fig. 1.—*A*, the condition of the tongue, Sept. 1, 1925, with a central open break in the surface which later healed over; *B*, present condition of the tongue with leukoplakia over the anterior end, especially.

linear arrangement were usually closely packed, side by side. Nevertheless, they had a fine, reddish demarcation from the next papules. In some places the reddish color between the rows of papules took on a somewhat grayish-red tint. As one observed the outer border of the areas described, the papules were not so evident. They were separated more widely from one another, and the area gradually fused into the normal skin. The demarcation from the normal skin was most marked above the clavicles on the sides, while down over the sternum it was more difficult to distinguish the involved area from the normal skin. Along the under surface of the upper arm, extending from the axillary space almost to the elbow, there were scattered discrete, brownish macules the size of a pinhead or smaller. There was no evidence, however, of any papular formation. Over each elbow the skin was slightly reddened. The skin was wrinkled somewhat, and a few keratotic papules were noted. Over the knees there was not so much of this wrinkling; there were numerous slightly reddened, raised papules which were follicular in type, some of them showing a fine, dark point in their centers. Both the palms and the soles showed a hyperhidrosis. There was a sug-

gestion of thickening of the skin over the palms. They were reddish, and there was a tendency to cracking of some of the folds of the digits. There was no thickening of the soles. The hands, up to the wrists, showed a reddish color, resembling an acrodermatitis chronica atrophicans. The skin over the dorsa of the digits had a suggestion of thinning and atrophy, and there was a definite anetodermia over the dorsum of the last joint of each finger. Fine parallel venules were scattered over the entire anterior surface of the pinna of the ears.

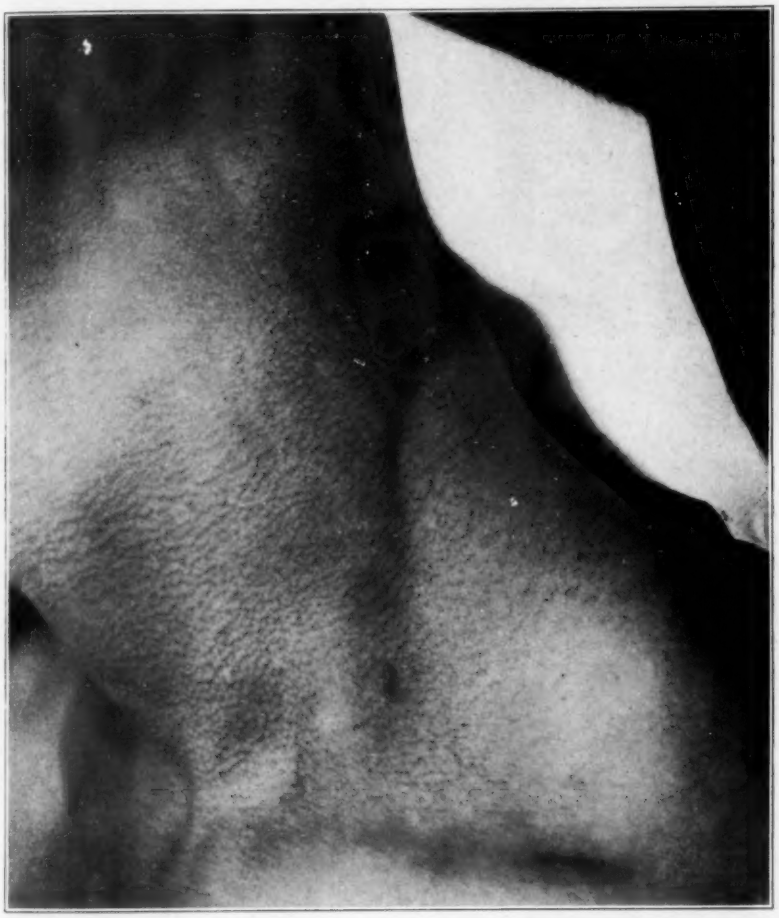

Fig. 2.—Left side of the neck, showing the arrangement of the papules with the reddish-brown pigment in between. Papules are arranged in rows following the somites.

On the heel of the right foot there was a bullous lesion which was broken. It was about 1 cm. wide and about 3 cm. long. No milia were noted in any areas.

Nails of the Hands: All the nails of the fingers and thumbs showed a condition which was almost similar. The base of the nail gradually fused into the skin with little or no sharp demarcation. The nails were smaller than normal, though the nail on the little finger of the right hand was somewhat thickened.

Fig. 3.—Dystrophic condition of the thumb nails.

Fig. 4.—Dystrophic condition of the finger-nails. The suggestive anetodermia over the dorsum of the last phalangeal joint should be noted.

They tended to converge somewhat like a pyramid, toward the distal end, and there was no free cutting margin, but here again the substance of the nail fused into the tissue of the skin. The nails showed raised, longitudinal grooves, the interlongitudinal portions being raised and plicated somewhat. There were a certain number of longitudinal cracks of the nails as well. The nail of the third finger on each hand was atrophic in type; the one on the left was notably so, having almost entirely disappeared.

Nails of Toes: The nails of the great toes approached normal more closely than those of any other digits. They had a free cutting edge, though the one on the right foot resembled more closely the description of the nail given for the hand. The remainder of the toe-nails had an appearance much like that described

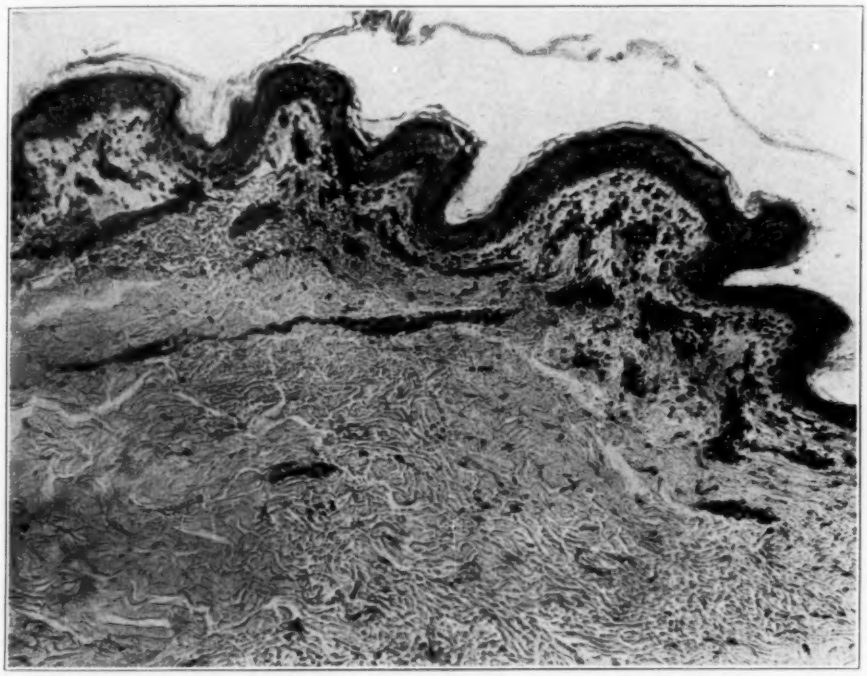

Fig. 5.—Area illustrating low epidermis, parakeratosis, dyskeratosis, dyskeratosis of prickle cells and dense clumps of pigment cells in the upper corium, along with some cellular infiltration, mononuclear in type, and some edema of the tissues. Hematoxylin and eosin stain; × 100.

for the fingers, though the nail of the third digit of each foot showed the tendency to a cone formation. The nails of the little toes were atrophic.

Further examination showed no abnormalities. The hair was of normal texture. Examinations of the blood gave negative results. Roentgen examination revealed no tumors.

Histologic Study of Tissue.—Specimens of tissue were taken from different areas on the patient's body. A small section was procured from the tongue but was unsatisfactory, and the patient refused to let us take any more. One portion of tissue was removed from a section of the neck from an area in which there was not such a prominence of the tubercles; another one was taken from a region

in which the tubercles were more prominent. The stains used were hematoxylin and eosin, van Gieson's stain, acid orcein, Mallory's eosin and methylene blue (methylthionine chloride, U. S. P.) and Mallory's phosphotungstic-acid stain.

Section Showing Less Marked Changes: The epidermis was low, with little or no papillary arrangement. It was only four or five cells in thickness. There was a slight amount of parakeratosis, up to two or three cells, and scattered here and there throughout the epidermis one noted dyskeratotic changes of the prickle cells. There was some slight increase in the vessels, especially just under the epidermis. The endothelial cells were somewhat swollen and the vessels enlarged with a little mononuclear cellular infiltration around them. Scattered, especially around the vessels, but noted in many different places in the upper corium, were numerous clumps of pigment cells. They were made up of dense, deep brown,

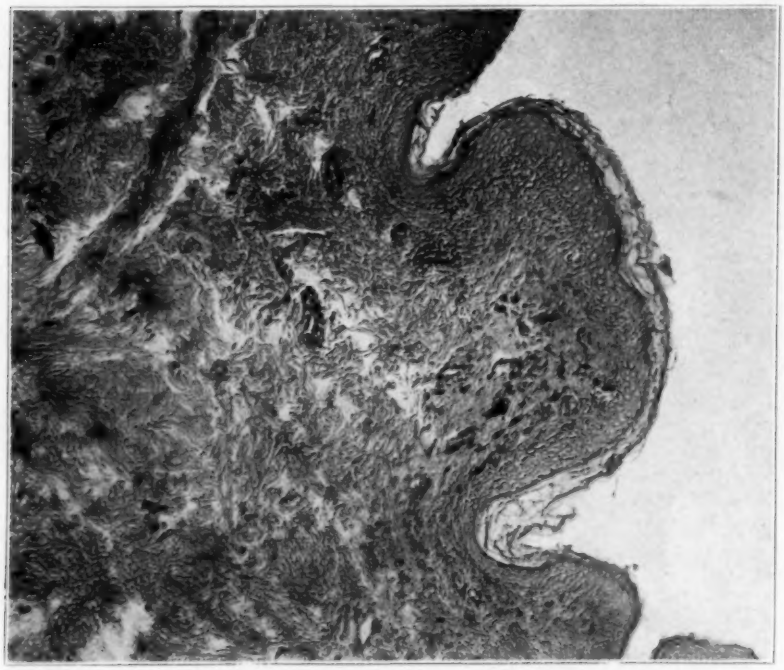

Fig. 6.—A portion of the tissue showing increased vascularity, swollen vessels, some mononuclear and cellular infiltration and edema around the vessels. Pigment cells in large clumps are to be seen. The epidermis is thin in the prickle cell layer. There is an increase in stratum corium and a dyskeratosis of the prickle cells. Hematoxylin and eosin stain; × 100.

closely aggregated pigment clumps. With Mallory's eosin and methylene blue one occasionally noted strands of pigment extending from these cells even up between the prickle cells of the epidermis. The pigment was noniron-bearing, as shown by the potassium ferrocyanide test. One occasionally noted the pigment cells deep down in the corium. The collagen fibers in the upper corium seemed to be somewhat shorter, and showed some hyaline change. There seemed to be no change in the sebaceous or sweat glands.

Section Showing More Advanced Changes: The epidermal changes in this section corresponded to those in the first section. The papillae were more marked, and one noted an extensive edema of the tissues of the papillae extending far down into the corium. The vessels were swollen with their endothelial cells likewise swollen. They were increased in numbers, and there was a mononuclear cellular infiltration around them; a few plasma cells and fibroblasts were also noted. The pigment cells were observed in large clumps, not only around the vessels but even around the hair papillae. Some hyaline change was noted in the collagen fibers of the upper corium.

A month after the Cleveland meeting, at a meeting of the Chicago Dermatological Society held Nov. 25, 1926, a case was presented by

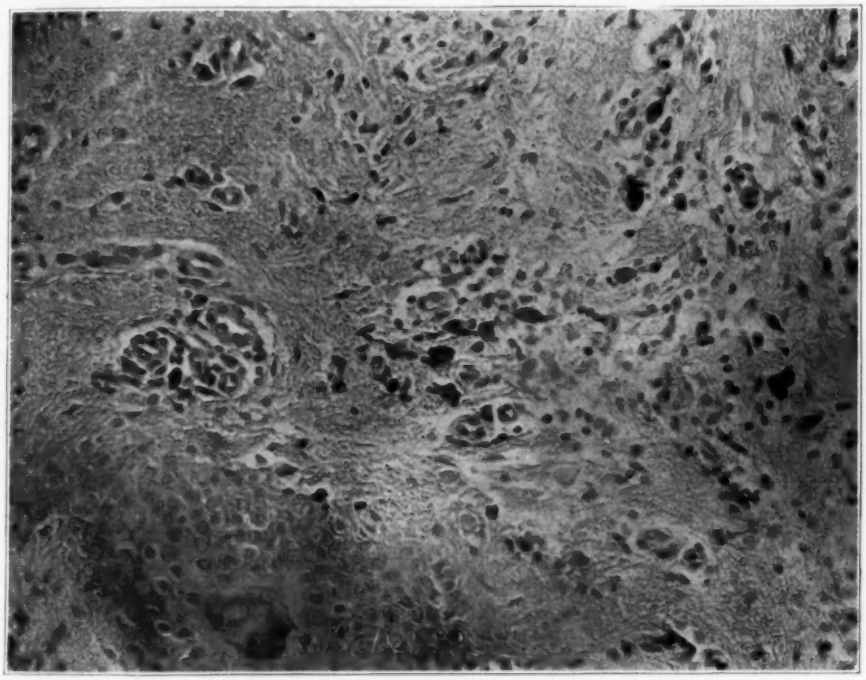

Fig. 7.—Area showing edema around the vessels which are dilated, swollen endothelial cells of the vessels and an extensive perivascular infiltration of mononuclear elements and some fibroblasts. Pigment cells are noted deep in the corium and to the right of the hair papilla. Hematoxylin and eosin stain; × 250.

Dr. Martin Engman [1] under the title of "A Unique Case of Reticular Pigmentation of the Skin with Atrophy." A description of the case follows:

History.—J. L., a man, aged 22, had been under observation for thirteen years. Since the age of 9 years he had had a peculiar condition of the mouth with an

1. Engman, Martin: A Unique Case of Reticular Pigmentation of the Skin with Atrophy, Arch. Dermat. & Syph. **13**:685 (May) 1926.

ulcer on the left side of the tongue and a spot of leukoplakia on the right side. No disturbances of the cutaneous surfaces were noted at that time. A brother, four years younger, had spots of leukoplakia on each side of the tongue. Bacteriologic examinations gave no information of any value. The patient was not seen again until November, 1925, when he had an ulcer of the leg.

Examination.—At that time he had a peculiar condition of the skin, unusual in the color of the pigment and its distribution. The color was of a gun-metal tint, bluish to purplish gray, and generally distributed over the body. Closely examined with a magnifying glass, it was seen that the pigment consisted of a peculiar reticular arrangement. There was a small white spot, 1 mm. in diameter, surrounded by pigment. The whitish spot did not always seem to be follicular. It was deepest on the abdomen and ran markedly in a reticular distribution. It was absolutely symmetrical in its placement, similar to a nevus. Scattered irregularly over the body, especially about the neck and face, there were large, oval, unpigmented, atrophic areas, from 0.5 to 3 cm. long and 1 cm. wide. The patient said that they were sites of former abrasions or infections. On the legs were large atrophic areas which were the site of abrasions. The condition of the hands resembled an acrodermatitis chronica atrophicans. The skin was atrophic, and the nails had disappeared. The same condition was observed on the feet. The skin of the scrotum and genitalia was mildly inflammatory, crusted, and presented atrophic areas. The patient had no eyelashes. The lids were somewhat everted and inflamed. There was some atrophy of the skin of the face, and it had the appearance of being affected by a mild form of a xeroderma pigmentosum, without freckles, black spots or keratoses. The mucous membranes of the tongue and cheeks presented whitened areas of leukoplakia. The patient was normal to every diagnostic test. There were no tumors as far as x-rays could show. The patient's brother was similarly affected. His condition was not so advanced. Histologically, the epidermis was thin and edematous from excessive serum. The rete pegs were flattened, and in places the epidermis consisted of only a few cells. The dermis was likewise edematous. Lymph spaces were wide open; the elastic tissue was normal. The collagen took the stain abnormally, and the upper part was granular. All the derma down to the level of the sweat ducts was full of pigment cells containing a brown, granular pigment which was iron-free. The sweat glands and ducts were normal.

In discussion, Dr. Francis Senear, Dr. James H. Mitchell and Dr. Edward Oliver of Chicago, all experienced dermatologists, observed that they had seen our case the month before and that it was an identical type of pigmentation. Dr. Senear made the observation that the dystrophy of the nails in our case was not exactly like this. Dr. William Allen Pusey, in discussion, said that he had seen a patient with a similar condition during the last few years, who had a pigmented area around the neck with punctate white mottling, which he took to be a congenital peculiarity. He thought the fact that the patient in Engman's case had a brother with a similar condition was highly suggestive of a congenital defect.

Though we have not seen Engman's cases, we are inclined, from careful study of the short report thus far available, to think that they

are similar to our case. The picture of atrophy is more pronounced in Engman's cases, but the changes in the nail correspond exactly. The peculiar whitish papule of the skin with the grayish-red zone around it—with, in our case, the reddish zone—likewise roughly corresponds. Both patients have a condition of the hands which resembles acrodermatitis chronica atrophicans. Moreover, both patients have symptoms of leukoplakia in their mouths. Engman's cases were familial and probably congenital. The condition in our case had been present as long as the patient and his family could remember. Again, the histologic picture seems to be similar in both cases—low epidermis, dyskeratotic prickle cells, numerous iron-free pigment cells in the upper corium, and extending deep down to the hair follicles and sweat glands; edema and cellular infiltration around the vessels of the upper corium; and swollen, shortened collagen fibers in the upper corium showing hyaline changes. We are therefore taking the liberty of studying Engman's case in connection with our own.

The history of age, the long duration, the dystrophy of the nails, the acrodermatitis chronica atrophicans appearance of the fingers, the pigmentation and the keratoses and leukoplakia of the mucous membrane of the mouth all suggest to us one of the congenital anomalies or dyscrasias. Probably the cases should be looked on as belonging in the group of the dyskeratoses.

In attempting to find similar examples of this unusual chain of effects, we have been more or less unsuccessful. Certain descriptions will, in some ways, correspond to ours and in others will differ materially. For example, some of the cardinal symptoms in our case have been reported in cases of Darier's disease, in erythrodermie ichthyosiforme congénitale, in epidermolysis bullosa dystrophica and in pachyonychia congenita of Jadassohn and Lewandowsky. On the other hand, there are some symptoms in our case which are foreign and not to be found in the diseases mentioned or in allied dyskeratoses. This naturally forces us to review and to discuss some of these conditions at some length. Possibly thereby we shall be better able to distinguish sharply defined groups of symptoms characteristic of this or that condition and also to determine whether they are in any way related to the condition in our case. We believe with Rothe [2] that as far as possible dermatologists should attempt to separate cutaneous effects into distinct groups rather than to jumble them indistinguishably together. We can all remember the time when "typhoid pneumonia" was a common diagnosis, and in our specialty it was not so long ago that the term eczema covered a multitude of evils.

2. Rothe, L.: Arch. f. Dermat. u. Syph. **102**:229, 1910.

In the hope of achieving this differentiation, or at least of making it easier to separate the dyskeratoses, Lenglet,[3] in 1903, in Brocq's clinic, enumerated the following main symptoms:

1. Agénésie or dysgénésie of the hair and nails (that is, the absence of effective function).
2. Functional disturbances of the sweat and oil glands.
3. Keratodermia palmaris et plantaris.
4. Exfoliation generalista (exfoliation, lamellar in type, of the new-born).
5. Hyperkeratosis typus: ichthyosis foetalis.
6. Hyperkeratosis typus: ichthyosis vulgaris.
7. Congenital ichthyosiform erythrodermia (Brocq).
8. Skin atrophies and deep atrophies.
9. Acanthokeratolysis congenital, bullae formation.
10. Epidermolysis bullosa—simple form.

To the "termes principaux" of Lenglet, Jadassohn [4] has since added three cardinal symptoms: pigmentations, anomalies of the mucous membrane of the mouth and anomalies of the teeth. He agreed with the Lenglet dictum that bullae are one of the best manifestations of congenital dermatoses.

It might be well to add another symptom not connected with the skin, but often seen in patients suffering from this type of dyskeratotic lesion, we refer to physical defects and to maldevelopments of the central nervous system (Klein [5] and Henrichs [6]). Moreover, there should probably be added a fifteenth symptom, as mentioned in the cases of Brünauer,[7] Burns,[8] Schäfer,[9] Thibierge,[10] Kreibich [11] and Hübner [12] and others—punctate to diffuse lesions over the corneas.

As can be seen from glancing over these fifteen principles in connection with the dyskeratoses, our case and the two cases of Engman have many of the symptoms in common. Many of these symptoms, however, are seen in greater or lesser degree with several of the other dyskeratotic diseases, and we believe it might be well to consider more fully some of them in differential diagnosis from our case.

Ichthyosis vulgaris is of itself and in its ordinary form a simple enough dermatologic condition. It has long been looked on as a familial

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5. Klein, Alfred: Arch. f. Dermat. u. Syph. **124**:571, 1917.
6. Henrichs, J., and Henriksen, P.: Norsk. Mag. f. Laegevidensk. **84**:1, 1923; abstr., Zentralbl. f. Haut- u. Geschlechtskr. **9**:106, 1924.
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11. Kreibich, C.: Deutsche med. Wchnschr. **34**:917, 1908.
12. Hübner, W.: Monatsh. f. prakt. Dermat. **24**:38, 1897.

disease. Henrichs [6] has called our attention to a series of ichthyotic families with mental disease. There were several generations in which the diseases appeared alternately or simultaneously. He stated the belief that they are due to primary ectodermal anomalies. Ideas in regard to its causation are various and need not be discussed here. However, as Bettmann [13] in his masterly monograph on "The Malformation of the Skin" well brings out: "If, in diagnosis, in addition to an ichthyosis vera, we consider clinically and histologically the border line and mixed cases in relation to anomalies of cornification and to certain nevi—under those conditions we sometimes have difficulties in differentiation." Bettmann (p. 724) says that "no case exists in which a hereditary keratoma of the palms and soles was present in a family of true ichthyosis or in connection with a true ichthyosis." Wolff [14] stated the belief that they are related. Oppenheim,[15] in discussion of a case of keratoma hereditarium palmaris et plantaris with symptoms resembling both an ichthyosis and the former disease, and in which two sisters had like symptoms, said that he felt that the differences between the two diseases were not sharp. He said that he saw few cases of keratoma palmaris et plantaris which did not show the effect of ichthyosis on the extensor surfaces. Kren thought the case shown was a pure ichthyosis and Riehl, an ichthyosis with atypical localization on the ulnar side of the palms.

Vörner,[16] in Riehl's clinic, thought that the two diseases should be sharply differentiated, likewise cornu cutaneum and ichthyosiform nevi. Waelsch [17] felt that the term nevus unius lateralis is not always appropriate, as it may involve more than the unilateral position, hence the term nevus systematisatus, i.e., involving a certain system. Some of these nevi have certain relations to ichthyosis in which we may meet all the transitions from a simple hyperkeratosis to an extensive papillary hypertrophy. He thought that we can bring out this peculiar relationship by the additional word "ichthyosiformis." Is not this an unfortunate term, however? Would not papillomatosis be preferable for, after all, it is not ichthyosis? There are cases called ichthyosis, however, which show many of the principles of Lenglet, for example, the case of Thibierge.[10] The patient was a boy, aged 12, who had had the eruption since the age of 13 days. All the cutaneous surfaces were the site of ichthyosis. There was an ichthyosis hystrix of the dorsal surface of the feet, hands and flexor surfaces of the body. The patient had the seborrheic type of

13. Bettmann, in Schwalbe, Ernst: Die Morphologie des Missbildungen, Die Missbildungen der Haut, Jena, Gustave Fischer, 1912.

14. Wolff: Arch. f. Dermat. u. Syph. **115**:1023, 1913.

15. Oppenheim: Arch. f. Dermat. u. Syph. **137**:63, 1921.

16. Vörner, Hans: Arch. f. Dermat. u. Syph. **56**:1, 1901.

17. Waelsch, Ludwig: Arch. f. Dermat. u. Syph. **114**:237, 1913.

head with alopecia of the head, ciliary and eyebrow areas. There was a thickening and folding of the mucous membrane of the buccal and nasal surfaces. Superficial opacities were noted on both corneas with radiating vessels. The palms and the soles were dry and showed numerous scattered cribriform depressions. The patient had a scrotal tongue and an opaline tint to the inside of the cheeks. The genital organs were infantile, the child was undeveloped and idiotic. Besnier called it an "ichthyosis paratypique." The case of Giovannini [18] is somewhat similar. A girl, aged 13, had had an eruption since birth. There was an enormous thickening of the palms and soles with cracks—ichthyosis hystrix—and folds with an ichthyosis serpentina of the rest of the body. On the upper part of the breast, around the neck and on the head, there was a peculiar appearance resembling gooseflesh arranged regularly in yellowish, slight excrescences, which Giovannini spoke of as ichthyosis anserina. It reminds us somewhat of the appearance of our patient's neck. They did not seem, necessarily, to have any connection with the hair follicles, but rather were hypertrophied sweat ducts. The nails were thick, up to 14 mm. in thickness, and were narrower and shorter than normal. They were not always parallel with the digits and had furrows over their surfaces in different directions. Their tops were rough and broken. There was a marked alopecia. There was a thickening of the epidermis over the palms and soles and enormous widening of the sweat ducts. Both cases should probably better be called examples of Brocq's erythrodermie ichthyosiforme congenitale instead of ichthyosis.

The erythrodermie congenitale ichthyosiforme of Brocq [19] has been carefully described in this country by MacKee and Rosen,[20] by Goeckermann [21] and by Wile.[22] The main symptoms are a redness of the skin, a marked hyperkeratosis resembling a high grade ichthyosis, a rich sebaceous secretion on the hair and face and a marked overgrowth of the hair and nails. One also notes hyperidrosis of the palms and soles and at times an alopecia, localized atrophies and changes of the mucous membrane of the mouth. Bullous lesions are a common concomitant but are not absolutely essential. Burn's case,[8] one much discussed in the literature, undoubtedly belongs here. It occurred in a boy, aged 16, with a generalized keratoderma, impaired vision from corneal opacities and deafness. There were keratoses of the palms and soles and eruptions in the folds of the joints. The mucous membranes were also involved. Over the face there was a reticulated, grayish,

18. Giovannini, S.: Arch. f. Dermat. u. Syph. **27**:3, 1894.

19. Brocq, L.: Ann. de dermat. et syph. **3**:1, 1902.

20. MacKee, G., and Rosen, I.: J. Cutan. Dis. **35**:235, 343, 511, 1917.

21. Goeckermann, W. H.: Bullous Ichthyosiform Erythroderma, Arch. Dermat. & Syph. **14**:158 (Aug.) 1926.

22. Wile, U. J.: Familial Study of Three Unusual Cases of Congenital Ichthyosiform Erythroderma, Arch. Dermat. & Syph. **10**:487 (Oct.) 1924.

symmetrical process with underlying erythema. Because of the dystrophy of the nails and the alopecia, instead of an overgrowth, Burns was not sure whether he should classify it under Brocq's description. Brocq [23] later wrote, however, "The adnexa of the epidermis, hair and nails can show all varieties of the development from hypertrophy even to atrophy." Undoubtedly the cases of Thibierge and Giovannini belong under this grouping rather than under ichthyosis. Here, also, would be placed the case of Goldsmith [24] and the one described by DuBois.[25] The main effect in this case was a marked onychogryphosis, leukoplakia formation in the mouth, pigmentary lesions and a papillomatous eruption most marked on the palms and soles, and on the latter location often accompanied by bullae. A family history of the condition is not uncommon, witness the case of Bettmann [13] and the two cases in children reported by Wile.[22]

Another of the congenital dyskeratoses which should be mentioned in passing is one already spoken of in connection with both ichthyosis and erythrodermie ichthyosiforme congenitale. We refer to keratoma palmaris et plantaris hereditarium. Balzer [26] reminded us that it was called "ichthyose locale" by Besnier. Alibert, on the other hand, called it "ichthyosis palmaris et plantaris cornée." It is true that it was not present in Engman's cases and that there was only a suggestion of the condition on the palms in ours; however, it is encountered in greater or lesser form so often with the other congenital dyskeratoses that it must not be overlooked. Family occurrence is common in this condition. It is generally seen on the palms and soles only and is accompanied by hyperidrosis. There are a few cases with lesions also on the back of the fingers and hands. Bettmann questioned the placing of Raedeli's case in this classification, as there were areas on the knees, over the shins and on the labia majora. Riehl,[27] in a discussion of a case of this disease said that he had seen the lesions not only diffuse, but also linear in type.

Neither Engman's cases nor our own show sufficient symptoms in common with the disease already mentioned to give us any help as to the diagnosis. Another of the dyskeratoses, however, possibly approaches a little nearer to our case—we refer to the dystrophic form of epidermolysis bullosum. Besides the bullae formation of the ordinary epidermolysis, we find in this condition atrophy of the skin and involvement of the nails. Milium may be found in both the simple and the dystrophic

23. Brocq, L., and Fernet, P.: Bull. Soc. franç. de dermat. et syph. **19**:327, 1908.

24. Goldsmith, W. N.: Brit. J. Dermat. **40**:59, 1928.

25. DuBois: Ann. de dermat. et syph. **6**:602, 1925.

26. Balzer and Deshayes: Ann. de dermat. et syph. **7**:386, 1906.

27. Riehl, in discussion on Brauer, August: Arch. f. Dermat. u. Syph. **114**:211, 1913.

epidermolysis bullosum. Bettmann gave the only example in the literature of the anomaly in four generations with the dystrophic form of the upper extremities and with the simple form of the lower extremity. Heredity is less common with the dystrophic form, and it is oftener later in appearing. Moreover, in the dystrophic form, the changes are usually seen on circumscribed areas—hands, feet, elbows and knees. Siemens [28] and Goldsmith [24] thoroughly discussed the entire subject of the bullous dermatoses. Siemens, from Jadassohn's clinic, stated the feeling that the term epidermolysis is often histologically incorrect and should be dropped for the more preferred term, bullosis mechanica, adding the term simplex, dystrophica or tarda for the respective type of the disease. We are inclined to accept the suggestion.

There is still another dyskeratotic process that should probably be considered at this point because of its confusing group of interrelating symptoms. In 1892, Brooke [29] reported under the title of "Keratosis Follicularis Contagiosa" what he thought had been an epidemic, in which he saw six children in one family and later three in another family on whom an eruption appeared on the neck, later on the arms, thighs and trunk, with papular, blackish spots which projected from the skin. He said that the skin was dry and that the papules showed a subacute inflammation; later, spines which rattled like scraps of metal as they fell and which left gaping plugs, could be removed from these papules. He said that there were some of them seen on the face but that the scalp was free. He treated them all with emollients and reducing substances, and they seemed to get well.

Morrow [30] had already reported a case which he termed "keratosis follicularis" associated with fissuring of the tongue and leukoplakia buccalis. This was observed in 1886. It was seen in a man, aged 21, and was said to have been of five years' duration. There was a grayish or brownish discoloration of all parts of the body except the face, palms and soles. The skin felt like the surface of a calf's tongue. The ducts of the sebaceous glands protruded and were occupied by round or comedo-like, grayish, dark substances with a spinous prolongation of from one-fourth to one-half inch in length. When pressed out, they were hard and dry and left a sticky substance at the base. In the mouth it was noted that the soft palate and the roof were studded with innumerable white, minute, brownish spots embedded or depressed, while the tongue, which was large, thick and flabby and rough, was rather whitish, pasty and deeply fissured. The buccal mucous membrane had an opaline or a bluish-white appearance, and there were thickened and raised furry, distinct tracts which were superficially fissured.

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28. Siemens, H. W.: Arch. f. Dermat. u. Syph. **139**:62, 1922.

29. Brooke, H. G.: International Atlas of Rare Skin Diseases, 1892, vol. 7, plate 22.

30. Morrow, Prince A.: J. Cutan. and Ven. Dis. **4**:257, 1886.

Little [31] presented two cases with similar symptoms before the London Dermatological Society. Barbe [32] reported the cases of two patients, aged 7 and 8, both of whom had congenital syphilis. The condition cleared up for a time under treatment with hydrargyrum internally administered but recurred promptly. Peyri-Rocamora [33] described an epidemic of something possibly like this condition in an asylum where fourteen children had a follicular eruption on the extensor surface, the folds of the joints and on the neck. This was made up of a hard cornification of the follicles, even hairy spines, from 0.5 to 1 or even 3 mm. high. They were a dirty white, yellowish or gray. There was some scaling. In some of the cases, there was also a perlèche. In some of the children, there were as many as from 50 to 200 elements. He thought that his cases were like Brooke's cases and quoted Brooke as having quoted Erasmus Wilson, who called it "ichthyosis sébacée cornée." Brooke quoted Hardy as having called it "acne sebacé." Lewandowsky [34] saw a man from India with an eruption of the extremities, extensor surfaces, back of the hands, gluteal regions, cheeks, ear and neck. The process was made up of numerous papular brownish lesions with a central horny mass which stuck out. He pressed this out, and it left a central depression. The skin between the lesions was normal. The corners of the mouth showed a high grade leukoplakia; otherwise there was nothing abnormal. Histologically, the process showed the "corps ronds" present, but he called it a keratosis follicularis Brooke-Morrow. There was no pigment, nor was syphilis present. He felt that in differential diagnosis one should consider a lichen spinulosis and Darier's disease. In the former disease there would be more group formation, while a keratosis follicularis is a diffuse process. Moreover, the latter is prominent on the extremities, while a lichen spinulosis is seen on the buttocks and on the back. Moreover, in lichen spinulosis all the lesions are of the same size, while in a keratosis follicularis they vary. He thought that it would be somewhat difficult to differentiate between this condition and Darier's disease. In Darier's disease there was more involvement of the inguinal regions, the genitalia, the abdomen, the head and the face. He did not feel that the "corps ronds" were specific for Darier's disease, that they could be seen under other conditions, though they were always present in Darier's disease. He said the French school called Brook's disease "acne cornée." He did not feel that it was a parasitic disease, and said that we should drop the word contagiosa and call it keratosis follicularis Morrow-Brooke. Schäfer [9] thought that the changes in this condition

31. Little, Graham: Brit. J. Dermat. **13**:417, 1901.
32. Barbe: Ann. de dermat. et syph. **2**:535, 1901.
33. Peyri-Rocamora, A.: Ann. de dermat. et syph. **3**:209, 1922.
34. Lewandowsky, F.: Arch. f. Dermat. u. Syph. **101**:5, 1910.

can never be called congenital and that they are easily influenced therapeutically. Whether, as Siemens mentioned, it is due to the action of poor soap, metallic dust (Blaschko), lubricating oil (Frieboes), petrolatum, paraffin or emery powder (Oppenheim [35]), or whether there is actually an excitant the removal of which causes healing, must be determined by further researches.

When Engman's case was shown at the Chicago Dermatological Society, the question was brought up by Pusey whether the process might be related to a juvenile type of acanthosis nigricans. The same point was made by Wile, who stated that the condition was unique. Wieder [36] recently reviewed the cases of this disease in connection with a case report concerning a girl seen at the ages of 10 and 13 in Wile's clinic. These cases have been seen as early as early infancy, at the age of 3 years and up to the age of 14 years in Neuman's cases. The process is essentially a pigmentation with keratotic hypertrophy. Ormsby [37] reminded us that there may be partial alopecia and changes in the nails with the condition. Reported lesions of the mucous membrane, however, are papillary or verruciform instead of the atrophic type as in Engman's cases and in our own case. Moreover, the parts ordinarily affected in this disease were exempt in our own case. Here again, while there are some points of resemblance, we believe that on the whole the cases of Engman and our case belong in a different group. Bettmann quoted Bogrow as placing the juvenile type of acanthosis nigricans (dystrophie pigmentaire et papillaire) nearer to Darier's disease or to atypical ichthyosis.

That Darier's disease has points in common with many of the congenital dyskeratoses is unquestioned. Rothe,[2] in 1910, and Spitzer,[38] in 1921, both from Jadassohn's clinic, have thoroughly discussed this subject. In this condition one may find characteristic longitudinal folds extending even down into the substance of the nail. The ends may be thickened and jagged. Moreover, changes in the mucous membrane have been observed. The family occurrence of the condition is too well known to mention. Pigmentations of the skin are present. Moreover, as in other congenital dyskeratoses, mental disturbances and anomalies are sometimes seen. Naturally, in this condition one has histologic changes that are so characteristic that in a given questionable case this in itself should be sufficient, if absent, to rule out Darier's disease. The follicular papular and later perifollicular papular eruption observed by

35. Oppenheim: Wien. klin. Wchnschr. **21**:260, 1921.

36. Wieder, L. M.: Acanthosis Nigricans, J. A. M. A. **87**:1964 (Dec. 11) 1926.

p. 495.

37. Ormsby, Oliver: Disease of the Skin, Philadelphia, Lea & Febiger, 1927,

38. Spitzer, Rudolf: Arch. f. Dermat. u. Syph. **135**:362, 1921.

Nicolau [39] in forty-four scorbutic soldiers would not be of a type to be considered in this paper. The process had no family relationship; it was seen in undernourished soldiers with scurvy and appeared as a papulokeratotic and even ulcerative eruption of the upper part of the extremities, abdomen, lower part of the heels and side of the torso. In some cases, the eruption was the premonitory symptom.

In 1905, Jadassohn and Lewandowsky [40] saw a girl, aged 15, with peculiar congenital changes in the nails. The nails were greatly thickened, were hard and had to be cut with a chisel. The finger-nails were long and narrow, smooth and shiny, rather transparent and, toward the tips, grayish. They were thick at their free ends and curved over transversely, like a sickle. The toe-nails were likewise thickened, especially that of the great toe, resembling an onychogryphosis. Growth was not especially rapid. There were no subjective symptoms in connection with the nails. The hair of the head showed no abnormalities. On the face, especially around the nose and the fold of the chin were red, pointed papules. On the nose there were vesicles with an alkaline reaction. There was hyperhidrosis of the nose, hands and soles of the feet. Generally in summer, rarely in winter, large bullae would form on callosities of the feet; there, too, the fluid from the vesicles was alkaline. Over the elbows and knees there were several grouped, slightly indurated, reddish papules the size of millet seeds. In their centers there was a not prominent hairy plug. The dorsum of the tongue was covered with an adherent, here and there, sharp and unequally bounded, whitish plaque. There were whitish strands on the borders of the tongue, some slightly raised. From time to time the patient showed an eruption on the scapulae and in the axillary spaces and gluteal region. There were irregular disseminated lesions, like the ones on the elbows and knees. The patient had a brother, aged 4, with a similar eruption.

Histologic study of a lesion with a horny cone showed a hyperkeratosis of the follicle and parakeratosis. The rete was thick and protruded deep into the cutis. The rete cells were vacuolated. In the cutis, there was a light edema and a little increase in cells. In a discussion of their case, the authors mentioned Darier's disease and keratosis follicularis Brooke. They did not feel that the symptomatology in either condition fitted that of their patients, and because of the prominent symptoms of the nail they suggested the term pachyonychia congenita. Their cases were presented by Lewandowsky [41] before the ninth Congress of the German Dermatological Association in 1907. As long ago as 1904

39. Nicolau, S.: Ann. de dermat. et syph. **7**:399, 1919.

40. Jadassohn, J., and Lewandowsky, F.: Ikonographia dermatologica **1**:29, 1910.

41. Lewandowsky, F.: Transactions of the Eleventh Congress of the German Dermatological Association, Berlin, Julius Springer, 1907, p. 333.

Wende [42] had reported the case of a child, aged 5, with symptoms somewhat resembling those already described. The condition began apparently when the patient was about 2 years of age. The child was bald from birth and showed comedo-like lesions over the head. There were bullae of the feet and later of the fingers, while the nails were thin and brittle and longitudinally striated. Both the hands and the feet showed a hyperhidrosis, while the finger-tips were atrophic, though x-ray pictures revealed no abnormalities. Naturally the query may be raised as to whether the condition was an epidermolysis bullosa dystrophia or bullosa mechanica dystrophica of Siemens; nevertheless, many of the symptoms coincide with those in the foregoing cases. Gaucher and Milian,[43] in 1905, reported a case with extensive keratosis palmaris et plantaris, agnesia of the hair, sudoriferous cysts with sebaceous contents, langue scrotale and leukoplakia of the mucous membrane of the mouth. The patient had a dry skin and pigmentation with keratotic lesions. At the tenth congress of the German Dermatological Association in 1908, Bettmann [44] showed a boy, aged 8, with lesions almost exactly like those in the patients of Jadassohn and Lewandowsky. He had identical changes in the nails, and there was a tendency to bulla formation on different parts of the body. The relation to epidermolysis bullosa seemed close; however, there was a leukokeratosis of the tongue and disseminated follicular keratoses of varying intensity. The patient also had a hyperhidrosis of the palms and soles and a granulosis rubra nasi. He had four brothers who were exempt from the disease. Bettmann (p. 735), in 1912, reported that he also had had as patients a father and three sons, the youngest 8 years of age, with similar congenital dystrophies of the nails, extensive leukoplakia of the mucous membrane and disseminated follicular keratoses of the skin. He also quoted Hartzell's case with leukoplakia and follicular keratoses and Babicek's with pachyonychia, symmetrical keratoses of the palms and soles, follicular keratoses and hyperkeratoses and deformities of the knees, elbows and teeth. In Dunn's [45] case, possibly questionable here, the patient had a keratosis palmaris et plantaris with alopecia totalis except for the top of the head. At puberty the hair was thicker on the head but never on the temples. There was a hyperhidrosis of the palms and soles, while the nails were thickened and the free edges broken—as in trichophytosis. The teeth were normal, and there was no family history. In Riehl's [46] patient, aged 23, there were callosities of the palms and soles, more

42. Wende, G. W.: J. Cutan. Dis. **2**:14, 1904.

43. Gaucher and Milian: Ann. de dermat. et syph. **6**:609, 1905.

44. Bettmann: Transactions of the Tenth Congress of the German Dermatological Association, Berlin, Julius Springer, 1908, p. 378.

45. Dunn, James: J. Cutan. Dis. **28**:191, 1910.

46. Riehl: Zentralbl. f. Haut- u. Geschlechtskr. **6**:335, 1923.

linear-like, and also callus-like lesions on the dorsa of the toes. There was a pachyonychia of the nails and a leukoplakia of the hard palate, cheeks and gingiva. The patient also showed keratosis pilaris of the calves; the joints were hyperflexible, and the patient had nervous manifestations. In Strandberg's [47] patient, the nails were thick and of a dirty gray color with smooth surfaces. There was hyperkeratosis with vesicles on the feet and callosities. The skin otherwise was normal. In Siemen's [28] patient, both the boy, aged 10, and the mother suffered from an eruption of the knees, elbows, buttocks and thighs, follicular in type, with a central blackish-brown, hairy plug. There were keratoses of the palms and soles, with sweating. The nails showed a pachyonychia and terrace formation of the ends. The nail-folds were a dirty black. In the axillae there were arsenical-like lesions. The tongue showed a light red hyperkeratosis and leukoplakia; leukoplakia appeared in the corners of the mouth. In Schäfer's [9] case, a boy, aged 12, showed light red to dirty brown skin on the knees, joints of the feet, elbows, nates and axillary folds. Situated on it were follicular keratoses with a central blackish-brown cone. On the left shoulder blade were two horny spinules 2 mm. long. The skin of the face was a bluish red, and the nose, palms and soles showed hyperhidrosis. Hyperkeratoses were present over the palms and the soles, making walking difficult. The walls were thickened and rolled in the form of an omega. They were blackish and were brittle at the ends. The tongue showed leukoplakia and in spots was bright red; a whitish plaque was also present on the inside of the cheeks. There were areas of alopecia areata, and the two middle upper incisors were dystrophic. The patient had a cataract of the right eye and incomplete descent of the left testicle. Brünauer,[7] in 1925, reported the case of a boy, aged 9, with similar symptoms and in addition a sickle-formed type of change on the cornea of the eye with keratitis. The patient had keratoses of the palms and soles, onychogryphosis, hairy formation on the limbs resembling systematized nevi and leukokeratosis in the mouth. In our country, Andrews and Strumwasser [48] reported at the 1928 meeting of the Dermatological Section of the New York State Medical Society, the case of a boy, aged 5, of low mentality, leukoplakia of the tongue, greatly thickened nails and a follicular eruption of the ankles, buttocks, lumbar region, forearms, elbows and knees. The boy at times developed waxy bullae of the feet. These authors thought that histologic study of their case showed bodies resembling "corps ronds" of Darier in the epidermis. There was no family history in the case. They also reported a case of Dr. Howard Fox which we do not believe contained sufficient evidence to be classified here.

47. Strandberg, James: Dermat. Ztschr. **22**:278, 1915.

48. Andrews, George C., and Strumwasser, Samuel; personal communication.

We have reported this group at some length, because we believe it to have a sufficiently definite chain of effects to justify the designation of a distinct entity—pachyonychia congenita as given it by Jadassohn and Lewandowsky. Andrews and Strumwasser were not inclined to put it down as a clinical entity but rather as an anomaly of Darier's disease, since they found bodies suggesting "corps ronds" in the epidermis in their case. Rothe, in 1910, emphasized the point that all Darier's cases will show the "corps ronds," but that one may find dyskeratoses in the epidermis in other dyskeratotic lesions that may resemble this one very much and yet not be Darier's disease. We believe their case to be a

Cases of Pachyonychia Congenita Described in the Literature Compared with Cases Reported by Us

Cases	Author	Follicular Keratoses	Circumscribed Plantaris et Palmaris Keratoses	Pachyonychia or Dystrophies	Leukokeratosis of Mucous Membrane of the Mouth	Palmar and Plantar and Nasal Hyperhidrosis	Vesicle Formation	Hair Anomalies	Congenital Tendencies	Changes in Eye	Changes in Teeth	Pigmentation	Atrophy
1-2	Jadassohn and Lewandowsky	+	+	+	+	+	+	—	+	—	—		
3	Bettmann	+	—	+	+	+	+	—	+	—	—		
4-8	Bettmann	+	—	—	+		—	—	+	—	—		
9	Babicek (nach Bettmann)	+	+	+	—	—	—	—	?	—	+		
10	Hartzell (nach Bettmann)	+	—	—	+		—	—	+	—	—		
11-12	Siemens	+	+	+	+	+	—	—	+	—	—		
13	Riehl	+	+	+	+	+	—	—	+	—	—		
14	Strandberg	—	+	+	—	+	+	—	+	—	—		
15	Schäfer	+	+	+	+	+	+	+	+	+	+	+	
16	Gaucher and Millan	+	+	—	—	+	+	+	:	:	:	+	+
17	Brünauer	+	+	+	+	—	—	—	+	+	—		
18	Andrews and Strumwasser	+	—	+	+	—	+	—	+	—	—	+	
19	Dunn	..	+	+	—	+	—	+	+	—	—	—	
20-21	Engman	+	—	+	+	—	—	+	..	—	—	+	+
22	Cole	+	?	+	+	+	+	—	—	—	—	+	+

true case of pachyonychia congenita. Schäfer, in a discussion of his case and in an attempt to place it, likewise felt that it was not a disease *sui generis,* but rather a systemic disease in the sense of a congenital dyscrasia. After all, however, as we look over this group of cases, we are impressed with the concordance of the symptoms. The condition is rare, and from 1905 to 1929, only eighteen true cases and one questionable one have been culled from the literature. After all, what is the study of medicine but an attempt to collect and separate groups of symptoms which seem to be most in accordance, and to attempt to set them up as distinct entities? It is only in that way that the fathers of medicine have been able to put its study and systematization on a scientific basis. We can remember the time when out of this same group of congenital dyskeratoses erythrodermie ichthyosiforme congenitale of Brocq was hardly accepted. We believe that pachyonychia congenita

is likewise entitled to a distinct niche, and as time goes on further cases will be seen, and the disease will become more firmly established.

Whether the cases of Engman and our own case should likewise be placed here or separated de novo is also difficult to say. Our cases present rather the picture of an atrophy of the skin. It was noted on the back of the last joint of the finger in our patient that the skin was suggestive of an anetodermia with acrocyanosis of the hands and feet. The enlarged parallel venules over the pinna of the ears were also unique. The symptoms were even more pronounced in Engman's one case of atrophic pigmentation. Moreover, this atrophic tendency was seen in the nails as well. In Engman's case they had disappeared entirely, and in ours they were stunted or dystrophic. The thickened nail is the cardinal symptom of cases of pachyonychia congenita; in fact, thereby the name is given. We remember Brocq's later statement in regard to "erythrodermie ichthyosis congenitale" that there could be all stages from a hypertrophy to a dystrophy in different types of the disease. If an attempt is not made to separate distinct groups having some symptoms in common, the question may properly be raised, why separate the dystrophic type of epidermolysis bullosa from the congenital ichthyosiform erythroderma? Or again, to go a little further, why separate Darier's disease or even keratosis follicularis Morrow-Brooke? It is only through endeavoring to classify the main groups of symptoms most closely related that we can render the study of dermatology clearer, more intelligible and more scientific. Bettmann (p. 731) has already expressly referred to the existence of such manifold combinations of congenital dyskeratoses: "they do not allow themselves to be easily arranged under the main types, but as mixed forms are especially important for the dictum that in all differentiation every type indicates a narrowly related whole. Why in a part of the cases pure single types are seen while in others very complex associations can be brought out with striking agreements is difficult to explain."

If we were to apply this dictum in our case, possibly we might consider it as belonging to the group of pachyonychia congenita. Moreover, Bettmann reported that in the father and three sons in his group of cases of pachyonychia congenita, there was a "Verkümmerungen," a stunting or dystrophy of the nails. Nevertheless for the present we are inclined to separate our case and the cases of Engman, though we have not seen them, into a distinct group of the dyskeratoses. Perhaps thereby others may pick out similar cases, or again some one later may feel that these cases should be better combined with the pachyonychia group in some other group. Purely as a provisional matter we would term them "dyskeratosis congenita with pigmentation, dystrophia unguis et leukokeratosis oris," more or less describing the main characteristics seen in the cases. That they are congenital we believe to be undoubted—witness

the histories. In one of Engman's cases, the condition was present at least from the age of 9, and as early as the patient could remember in our case. It is also familial, at least it was so in Engman's case. If Bettmann's four cases were to be placed here, this would be more evident. The main symptoms are those of dystrophy or atrophy of the nails, a peculiar pigmentary eruption of the neck and upper part of the chest in one case, and almost universal in Engman's cases, a dyskeratotic eruption of the elbows and knees, an acrocyanosis of the extremities and ears and a leukoplakia of the mucous membranes of the mouth. "The hands looked like acrodermatitis chronica atrophicans." The process is not of a type that could be classified under the ichthyoses or the keratosis palmaris et plantaris or epidermolysis bullosa (bullosis mechanica simplex, dystrophica, or tarda). It would hardly fit in the Darier's complex or with the keratosis follicularis—Morrow-Brooke. The symptoms might possibly be nearer to the juvenile type of acanthosis nigricans, but unfortunately the areas usually involved in that disease were free from lesions in our cases, and the papillomatous character of the disease was entirely absent. We believe the effect corresponds closest to pachyonychia congenita of Jadassohn and Lewandowsky, eighteen definite cases of which have thus far been described. Because of the atrophic or dystrophic type of our cases, we would separate them as still another related group and possibly as a distinct entity.

What the cause of these allied congenital dyskeratoses may be is a great question. They are often familial. Henrichs would say that the cause was a pathologic disposition of the ectoderm. Consanguinity undoubtedly plays a rôle in certain of these cases, as has been brought out in a family whose cases have been reported from Jadassohn's clinic. Siemens stated that ichthyosis vulgaris is a dominant, and ichthyosiforme congenitale erythrodermie a recessive, characteristic. Bettmann (p. 715) says that the manner of separation of the epitrichium may be of importance in all anomalies of keratinization. Lenglet expressed the same feeling. Meirowsky, according to Fischer, thinks keratoma palmaris et plantaris is a dominant characteristic. He thinks that the anomaly of the hair, nails, palms of the hands and soles of the feet is a disease of the skin in which single phenomena are coordinated and are to be conceived as a destruction of the germ plasm and inherited as a dominant character of the general germ plasm, the ectoderm.

CONCLUSIONS

1. A report is made of a case, and what seem to be two similar ones from another clinic, of a unique dyskeratosis of the skin.

2. The condition is characterized by being probably congenital and familial and is accompanied by symptoms of atrophy, pigmentation, dys-

trophic changes in the nail and by leukokeratosis of the mucous membrane of the mouth.

3. The process has some characteristics in common with the dystrophic type of epidermolysis bullosa (bullosis mechanica dystrophica), with Darier's disease and with the juvenile type of acanthosis nigricans. It probably approaches closest, however, to the syndrome described by Jadassohn and Lewandowsky under the term pachyonychia congenita.

4. We believe the characteristics of the latter syndrome to have been well enough defined and described in eighteen cases and one questionable case, which we have culled from the literature, to entitle it to the designation of a distinct disease.

5. In the three cases mentioned, the condition appears to be most like pachyonychia congenita, but because of the dystrophic effect we would provisionally suggest the name of dyskeratosis congenita with pigmentation, dystrophia unguis and leukokeratosis oris.

In conclusion we wish to express our thanks to Dr. Maurice L. Richardson and to Dr. Otto Saphir of the Pathology Department of the Cleveland City Hospital for their help in the preparation of pathologic specimens and in the making of photomicrographs.

ABSTRACT OF DISCUSSION

DR. MOOK, St. Louis: I think unquestionably the cases shown by Dr. Engman and Dr. Cole belong to the same group. In our case the boy was 12 years of age when we first saw him. At that time he had ulcers only on both sides of the tongue. The skin, hair and nails were normal. An attempt was made to diagnose the condition tuberculosis, but he had no visceral lesions of tuberculosis. We next saw him ten years later, and he had then developed the universal condition of the skin. His hair was thin, the eyelashes and eyebrows had disappeared, the nails were completely destroyed and he had pigmentation of the mucous membrane with considerable atrophy of the mucous surfaces of the cheeks. At the same time a younger brother was developing a similar condition on the tongue, and we believe that in time he will develop the same condition. In this family, at any rate, the condition is familial and probably congenital.

There was one feature about our case that was suggestive that it might belong to the nevus group, in that on the abdomen there were scroll-like lesions similar to those in nevus unius lateralis, without the keratosis. In addition, there was a line running down from the sternum to the pubis and a cross line at about the tenth ribs, so that his trunk was divided into quadrants, the upper left and the lower right being much more pigmented than the upper right and the lower left. The general health of the patient was unaffected, but the process is progressive.

DR. HOWARD FOX, New York: I was much interested to hear of this case of Dr. Cole's. It did not seem to be precisely like the one reported in the *Ikonographia* by Jadassohn and Lewandowsky as pachyonychia congenita. As Dr. Cole mentioned a case of mine and put it in the doubtful class, it might be well to discuss it. Dr. Andrews and Dr. Strumwasser who reported a case from New York considered my case almost identical with their own. The nails were thickened rather than atrophied and were precisely like the picture published in the *Ikonographia*. The

patient was a boy, about 4 years of age, who had wartlike lesions on the extremities and buttocks and a few flaccid bullae on the feet underneath callosities. He did not have leukokeratosis of the tongue, nor did he have lesions suggesting granuloma rubra nasi, but it seemed to me that it was unquestionably a case of the anomaly described by Jadassohn and Lewandowsky.

PROF. JOSEF JADASSOHN, Breslau: Dr. Cole was kind enough to show me his interesting case when I was in Cleveland. I agree with him that it is to be classified as one of the congenital anomalies, and would like to add only a few remarks.

As Dr. Cole noted, Lenglet was the first to enumerate the different symptoms which are to be found in this group. Dr. Cole and I completed this series. I want to call attention to a further symptom, namely, hyperhidrosis of the fingertips while the palms are dry. I saw this symptom in different forms of this group, even in common ichthyosis.

The well known types of congenital diffuse anomalies of the skin are characterized by typical combinations of some of these single symptoms. If this combination is a different one, then we have the atypical cases, as, for instance, Dr. Cole's case. It seems to me important to describe these cases exactly as Dr. Cole did, because they may not be as rare as our literature seems to show, and if we have no exact description, we cannot bring together the corresponding cases. This is necessary not only from a diagnostic standpoint but also for studies about the heredity of single diseases. It seems important, especially in these atypical cases, to find out whether the same atypical combination is to be seen in different members of the same family. We must always try to examine as many relatives as possible, to establish whether or not there is consanguinity of the parents, and so forth.

I want to emphasize that there are in this whole group many abortive forms ("formes frustes"). They are well known in the common ichthyosis, but as far as I can see they are not recognized, e. g., in the cases which belong to the ichthyosis paratypica of Besnier or the hyperepidermotrophie of Brocq. I see, not infrequently, stripes of hyperkeratosis on the flexor sides of the knees and the elbows, on the front side of the axillae and on the navel. I have seen that anomaly sometimes even in several members of the same family. It seems to be not without interest that many of the single symptoms of these congenital anomalies are only exaggerations of qualities of the skin which we generally do not call anomalies. I mention hyperhidrosis, keratosis pilaris, dry and somewhat hyperkeratotic skin, etc. In this connection I must mention the dilatation and light hyperkeratosis, and later on, atrophy of the follicles of the neck and chest, and the form of the toe nails, which resembles the pachyonychia, by their strong convex (but not hyperkeratotic) forms. Perhaps it will be possible by studying these formes frustes in families to find more material for the study of heredity in skin diseases.

Finally, I want to say that the name pachyonychia described by Lewandowsky and myself is incomplete. We wanted to publish in the *Ikonographia* the results of studies on the nail form, but it will be necessary to complete this name, and we can do this only by a short enumeration of the principal symptoms. In this manner it will be necessary both to give to the new cases the right place in the group of congenital anomalies, and to individualize it in such manner that it will not be lost in the future.

DR. WILLIAMS, New York: I am much interested in this presentation for several reasons. In the first place, I am interested in the diagram showing groups of circles and straight lines, which I suppose are intended to show some sort of relationship. It is a vivid picture, satisfying to the eye, but misleading for that reason. It seems to clarify our knowledge, but really does nothing of the kind, because there is no definition of the exact relationship which is intended.

In the second place, we have a good deal of confusion because, as Professor Jadassohn has brought out, we have the occurrence in a family of a disorder with definite but varied symptoms. All the cases have certain points of resemblance, but there are many individual differences. The only conclusion, I think, is that there is some underlying fault, some dysfunction of the elements that control development.

I wish to emphasize further what Professor Jadassohn said, that if such cases are to be studied, all members of the family must be studied, and all the symptoms recorded.

The deformity of one of the nails looked, as Dr. Cole said, exactly as if it had been caused by trichophytosis. It certainly did. I have seen many of these, in which I was unable to decide whether the deformity was due to dystrophy or to an infection caused by *Trichophyton*. I believe that in many cases dystrophy, whether produced by injury, by malnutrition or by some other causes, is responsible for the greater part of the visible deformity, and furnishes a suitable ground for the growth of various organisms, including the trichophytons.

DR. COLE, Cleveland: In reply to Dr. Williams' question, I may say that at one time we wondered about the possibility of this being a parasitic process. We thought we cultivated a thrushlike organism from the tongue, and looked for it zealously in the nails. I think it is possible for a parasitic organism to get in and yet mean nothing.

I did not emphasize, possibly, as much as I should the fact that this is undoubtedly a familial disease. In the cases reported by Bettmann the father and three sons had the condition. In the report from the Jadassohn clinic there was a marked influence of consanguinity when cousins married, the flare-up in the next generation illustrating this. In our case we found no family history in this way as far back as we could go. I have not seen the case mentioned by Dr. Fox. I saw the reprint but was not sure about putting it in this group, or whether it was a type of epidermolysis bullosa. Of course, Dr. Fox saw the patient and knows far more than I could get out of the report.

As far as the circles in one of our illustrations are concerned, any one who reads anything from Brocq's clinic knows that the clinicians there are fond of circles, and this illustration was from his clinic.

THE SKIN IN DIABETIC PATIENTS

FURTHER STUDIES *

ARTHUR M. GREENWOOD, M.D.
AND
ETHEL M. ROCKWOOD, M.D.
BOSTON

In view of the probable increased susceptibility to infection of the feet generally considered to be present in diabetic patients, we have attempted to ascertain the bacterial and fungous flora of the interdigital spaces of the feet in a series of patients taken as they were admitted to the New England Deaconess Hospital. One hundred patients, whether or not they complained or gave a history of disease of the skin in this location, were examined, and it seemed to us that these patients might be considered fairly representative. Scales and bits of skin were taken from the interdigital spaces, collected in sterile containers, examined microscopically and later were planted on Sabouraud medium. There was no cleansing of the skin in any way before taking the scales and no treatment of the scales before planting, since we wished to obtain all organisms which might be present.

Fifteen plants were made of the original scales in each case, and transplanted to special mediums as necessary for further investigation. Bacteria were grown in hormone broth, blood agar and blood serum, and classified according to their hemolyzing and pigment-producing qualities. Of the 100 patients examined, seventy gave clinical evidence of fungous infection and thirty gave negative results. This incidence corresponds closely with figures previously reported.[1] Seventy-five per cent of those examined clinically were in the age groups from 40 to 70, and of those who gave positive reactions 80 per cent were in these age groups.

The average blood sugar content of the group was 205 mg. per hundred cubic centimeters; that in cases in which the results of examination were positive, 200 mg. per hundred cubic centimeters, and that in the cases in which the results of examination were negative, 210 mg. per hundred cubic centimeters. Fifty-seven, or 71 per cent, of the seventy

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* Submitted for publication, April 1, 1929.

* Read at the Fifty-Second Annual Meeting of the American Dermatological Association, San Francisco and Del Monte, Calif., July, 1929.

1. Hulsey, S. H., and Jordan, F. M.: Ringworm of the Toes as Found in University Students, Am. J. M. Sc. **169**:267 (Feb.) 1925. Greenwood, Arthur M.: A Study of the Skin in 500 Cases of Diabetes, J. A. M. A. **89**:774 (Sept. 3) 1927.

cases in which the results of examination were clinically positive gave microscopically positive results. This is a considerably higher percentage than we have ever obtained before and is due, we believe, to more careful and persistent search in each case. The result of the cultures is given in table 1.

Beyond convincing ourselves that the fungi belonged to the pathogenic group, further identification has not as yet been completed, since that did not seem essential to the purpose of this investigation.

The fact that so few saprophytes, if *Aspergillus* and *Pencillium* may be so called, and yeasts were found is interesting and indicates that they

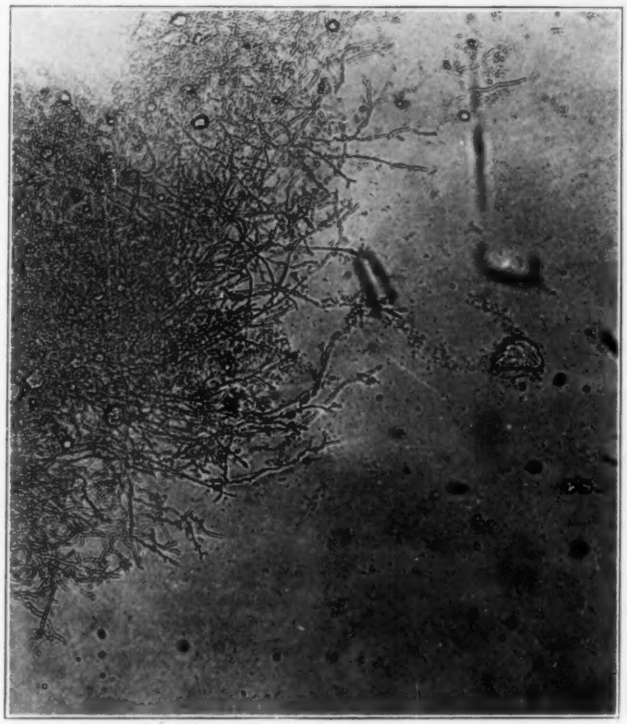


Fig. 1.—Low power photomicrograph of extemporaneous preparation of the nail.

are not common inhabitants of the interdigital spaces. This might also be taken as supporting the view that when yeasts are repeatedly found in dermatoses, they may fairly be considered as an etiologic factor in these conditions.

In twenty-four cases in the series, specimens of opaque and deformed nails were examined; 70 per cent of these microscopically showed actively growing mycelium comparable in all respects except

one to the mycelium found in scales. This difference consisted in the fact that some of these fungi showed the development of conidia, chlamydospores and fuseaux in the nails.

We have not as yet obtained cultures from these particular patients so that we may find them to be different from the skin pathogens. They so closely resemble the skin fungi in all other respects, however, that we believe that their cultures will prove to be similar. Forty-two per cent of these patients whose nails were involved showed no evidence of

Fig. 2.—High power photomicrograph of same preparation as in figure 1, showing resemblance to figure 4.

fungous disease of the skin but showed clinical evidence of infection of the nails, and 87 per cent of these cultures from the nails were positive. This observation, in addition to the possible production of fruiting bodies in the nails, bears out the suggestion made by Dr. C. M. Williams that this may be an active and important source of reinfection of the skin. As 70 per cent of the patients showed both clinical and microscopic evidence of infection of the skin, and as nearly 50 per

TABLE 1.—*Flora of Interdigital Spaces*

	Number	Per Cent
Fungi—pathogenic	9	13
Fungi—saprophytic	2	3
Yeasts	4	6
Staphylococci	70	100
Streptococci	1	1.4

Fig. 3.—High power photomicrograph of the same preparation as in figure 1, showing resemblance to figure 4.

TABLE 2.—*Bacterial Flora of Interdigital Spaces*

	Per Cent
S. albus	61.5
C. aureus	33.5
S. citreus	3.5
Streptococcus	1.5

cent of those apparently without an infection of the skin showed an infection in the nails, it almost seems that we are justified in considering fungous infection of the feet as universal.

In regard to the bacterial flora it is evident that staphylococci are invariably present and in the proportion shown in table 2.

The degree of hemolysis for each culture has been worked out, but for the purposes of this paper it does not seem necessary to include it. Only 13.2 per cent of all the cultures were nonhemolytic. The purpose of this particular part of the investigation is to determine whether any

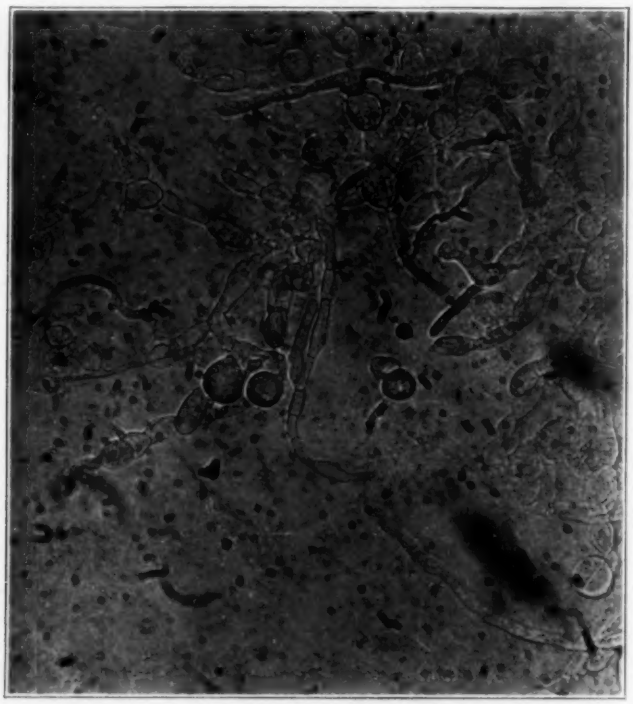


Fig. 4.—Culture of Epidermophyton cruris.

connection can be traced between the bacteria ordinarily found on the skin of a diabetic patient and those recovered in the definitely septic conditions encountered.

All diabetic patients admitted to the Deaconess Hospital are carefully instructed in the care of the feet, and these instructions are demonstrated on each patient in the Foot Beauty Parlor where there are several experienced nurses and a chiropodist in attendance. We attempted to check up on the value of this instruction by recording the number of those admitted for the first time and the number of those previously admitted who gave positive or negative results on examination. We found that 34 per cent of the patients who were

examined before were still infected and that 66 per cent of those who were admitted for the first time were infected, so that there is considerable value apparently to such instruction.

In October, 1927, Folin, Trimble and Newman [2] published an article on the distribution of dextrose injected into animals. They showed that:

> One important outlet for the sugar as it leaves the blood is the skin. The skin is not a storage place for glucose or glycogen, for in a fasting condition it contains practically no glycogen and only a trifle more sugar (about 10 mg. per cent)

Fig. 5.—Beginning of fragmentation of mycelium.

> than skeletal muscle; but as a temporary receptacle for sugar in response to sugar injections, the importance of the skin seems to us to be very much greater than has been appreciated before.

As these authors stated, when the blood sugar level is rapidly raised there is a rapid distribution of sugar into the skin, the content of which may reach from 300 to 500 mg. per hundred cubic centimeters when the blood sugar is from 600 to 700 mg. per hundred cubic centimeters. As

2. Folin, Otto; Trimble, Harry C., and Newman, Lloyd H.: The Distribution and Recovery of Glucose Injected Into Animals, J. Biol. Chem. **75**:263 (Oct.) 1927.

the blood sugar level falls below that of the skin, the sugar diffuses back into the blood stream. It seemed to us that this might offer an explanation of the apparent high incidence of diseases of the skin and infections in diabetic patients, and we attempted to correlate observations on the blood sugar with the occurrence of infections in a series of 100 diabetic patients whose blood sugar concentration had been recorded over a period of at least five years. The results, however, were entirely disappointing. In the first place, only 5 per cent of the patients had suffered from infections during this period, although the average blood sugar con-

Fig. 6.—Fragmented mycelium, apparently intercellular.

centration of the group was 198 mg. per hundred cubic centimeters. Second, it was seen that so many unknown factors such as diet, amount of insulin taken, etc. entered into the problem that the results were wholly unreliable. The result strengthens our impression that hyperglycemia in itself cannot be considered the important factor in this problem. It may also be said that in our investigation of the flora of the interdigital skin, similar attempts to correlate high blood sugars with active fungous or bacterial infection were entirely negative. No correspondence could be found between high sugar concentrations and active infection.

Fig. 7.—Further degeneration, showing loss of linear arrangement.

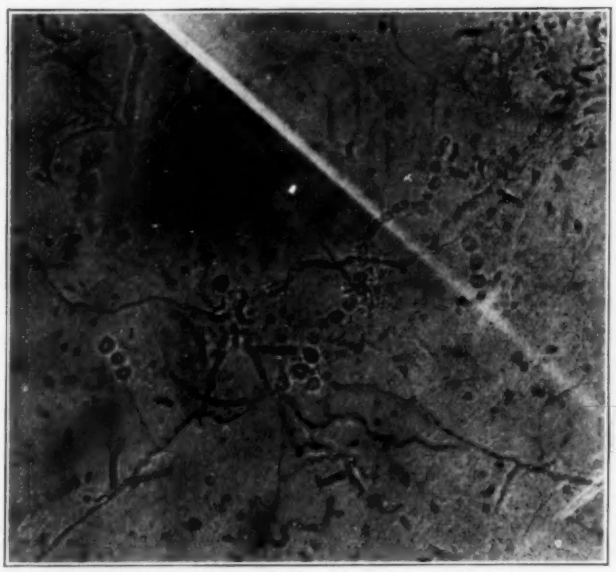


Fig. 8.—Continued degeneration. Some fragments showing angular borders.

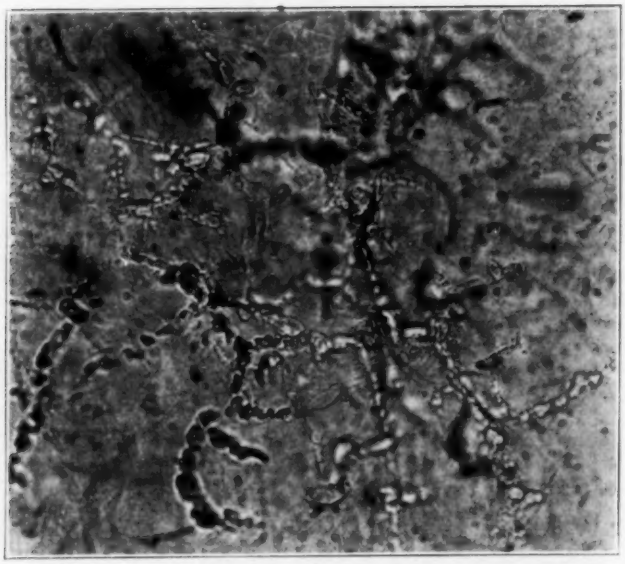


Fig. 9.—Fragments swollen and with irregular contours. In the same field, fairly normal fragments still remain.

Fig. 10.—Last stage of degeneration.

In regard to the microscopic examination of scales for fungi we have become convinced of the identity of the somewhat bizarre, degenerate, "mosiac" forms with the usual actively growing mycelium. We have repeatedly been able to demonstrate the continuity of these forms with normal mycelium in the same scale. It is, however, extremely difficult to show this continuity in photomicrographs because of the different levels in which the mycelium may grow, but we firmly believe that scales, even though showing only the "mosaic" forms, should be classified as positive. The series of photomicrographs shows the mycelium in extemporaneous preparation of scales. These are arranged serially to correspond with what has seemed to us to be the various stages of degeneration.

CONCLUSIONS

1. Seventy per cent of the feet of diabetic patients show pathogenic fungi; 100 per cent show the presence of staphylococci. Yeasts and saprophytic fungi are rare.

2. It is impossible at present to correlate hyperglycemia with these or other local infections.

3. We believe that all thickened and opaque nails on the feet will show fungous infection.

4. Instruction in the care of the feet has improved the condition of the feet 50 per cent.

ABSTRACT OF DISCUSSION

DR. WERNER JADASSOHN, Zurich: In their interesting investigations the authors found no connection between the quantity of blood sugar and the presence of parasitic fungi. Moreover, it is not certain that the frequent presence of the fungi has anything to do with diabetes of the patients, because a high percentage of mycosis of the feet is found in otherwise healthy people. In spite of this, it seems to me that it is not possible to deny the importance of diabetes for mycotic skin diseases. At least in all intertiginous oïdiomycotic diseases a connection with diabetes seems to me to be certain. The number of diabetic persons among patients suffering from oïdiomycosis is relatively so large that chance is entirely excluded. The course of the diabetes is important for the course of the oïdiomycosis. I remember a case in which the oïdiomycosis always disappeared after injections of insulin and reappeared quickly after the injections of insulin were discontinued. This phenomenon was experimentally demonstrated in animals in the Breslau clinic by Biberstein, in dogs, after the removal of the pancreas.

DR. E. L. MCEWEN, Chicago: I have read Dr. Greenwood's paper with great interest, and when he closes, I wish he would give us some information about the "foot beauty parlor" he mentions. I judge it is connected with the hospital, and perhaps some further information regarding it would be valuable to all of us.

DR. E. D. CRUTCHFIELD, San Antonio, Texas: It occurred to me that it might be of interest to know what percentage of nondiabetic patients in that

locality show pathogenic fungi. I think that locality has much to do with this. A few years ago, as a member of a commission to study the state prisons in Texas, we found that from 83 to 85 per cent of the prisoners had fungi infections. This may be due to the heat and dampness of the Gulf Coast, but in Texas I am sure 90 per cent of the inhabitants have fungi which can be cultivated in the way Dr. Greenwood mentioned. I wonder if the nondiabetic subjects do not show about the same percentage of infections as the diabetic patients.

DR. CHARLES MALLORY WILLIAMS, New York: I cannot keep still when any one talks about ringworm. Not only the presentation but the article itself has interested me greatly. Dr. Greenwood reports finding clinical evidence in 70 per cent of his cases, and proved this in a large percentage. That is a good result, and no doubt it is due to care. I wish to confess my inability to tell where to look for ringworm organisms. A patient was seen at the hospital with a typical tinea of the feet, so reported from the laboratory, and she also had trichophytid. I sent her to my office in order to confirm the laboratory observations. I examined many scales and vesicle roofs, but found no parasite. I was about to give up when the patient told me I was not looking in the right place, and that if I looked where the physicians at the hospital did, I probably would find what I was looking for. She said that at the hospital scrapings were taken from the wet places on the inside of the toes. I did this and found the scrapings alive with micro-organisms, where I had not expected to find them. It is hard to tell what the good hunting grounds are.

Dr. Greenwood did not report, so far as I am aware, whether the apparently normal feet were examined microscopically. This I think is important. I think that in these reports we should confine ourselves to those cases in which the microscope or test tube has been used. I think that we cannot tell without these aids. The symptoms may be produced by a bacterium or by a yeast. I agree with Dr. Weidman that we should make a diagnosis of intertrigo until we can use the microscope.

A few years ago I reported before the society on finding *Epidermophyton* on normal toes. I never again will report what I find on normal skin. I do not know what the "normal" skin is. We should always say that the skin looks dry, smooth, and so on. I do not remember what the skin looked like in that case I reported. There may have been a little scaling or dryness or maceration. When one says the skin, or any other organ is normal, he really means that there are no lesions which he has yet learned to differentiate.

DR. WILLIAM H. GUY, Pittsburgh: It seems to me perhaps not unreasonable to believe that in view of this work, particularly coupled with Folin's work showing a high percentage of sugar stored in the skin that the increasing number of fungus infections we see may be partly accounted for by the dermal increase in sugar. I am sure we all have had the experience of having cases in which the condition was resistant at times, and flared up for no apparent reason while the patient was under treatment, and it occurs to me that this may be the explanation. I was interested after reading Folin's work to hear an Indian physician say that he could have saved a year's time by studying in India, for there the urine of suspected diabetic persons is poured on an ant hill and, of course, in the presence of sugar, the activities of the ants establish the diagnosis. It was thought that had Folin placed his experimental animals on ant hills—the selection of sugar saturated tissues might have solved the problem. Of course, ants may not care for sweetened proteid.

DR. ARTHUR M. GREENWOOD, Boston: I was much interested in Dr. Jadassohn's statements. Our results had to do with the incidence of the disease, and it did not seem to me that the difference in incidence between this disorder in our

locality and others was sufficient to say that diabetes was accountable for the increase.

Regarding the "foot beauty parlor," as you know, most diabetic patients die not of diabetes but of complications, and most frequently of surgical complications. The most frequent of these are in the feet and legs, and Dr. Joslin arranged what he called a "foot beauty parlor" where his patients are taught how to take care of the feet. Two trained nurses and a chiropodist are in attendance, and all patients are shown how to take care of their feet for the rest of their lives. This instruction apparently cuts down the fungus infection of the feet about 50 per cent in old cases.

Replying to Dr. Williams, the normal feet were not microscopically examined at the clinic.

Biography

ALFRED HARDY

1811–1893

B. BARKER BEESON, M.D.
CHICAGO

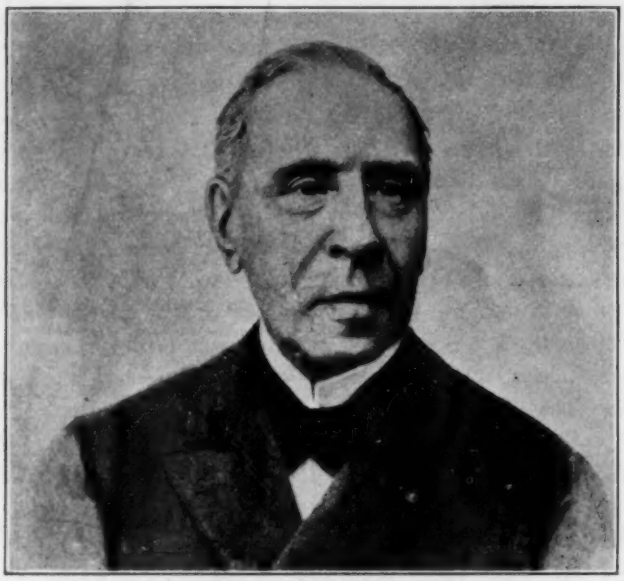
Like many other illustrious French physicians, Louis Philippe Alfred Hardy, born Nov. 30, 1811, was a Parisian by birth. When 21 years old he became an intern in Paris, and he was afterward fond of relating that Bazin and he had been fellow interns at Hospital Saint Louis in 1835. Hardy's thesis for the doctorate in medicine (1834) was entitled, "The Use of Caustics in the Treatment of the Affections of the Cervix Uteri." Rostan and Andral are said to have been his favorite teachers, but so far as dermatology is concerned Hardy is considered to have been a pupil of Alibert. In 1839, Hardy became Physician to the Hospitals of Paris, and eight years later he was successful in the concours for professeur agrégé, choosing internal medicine as his field of endeavor. Hardy's hospital career was a lengthy one, lasting forty-eight years. He passed almost one half of this time at Hospital Saint Louis and eleven years at the Charité Hospital. He became interested in skin diseases soon after coming to Hospital Saint Louis in 1851, and two years later he began lecturing on them. At first his course was a free lance affair, but later he was officially delegated to teach dermatology. He was a teacher of dermatology for more than twenty years. In 1867, the two chairs of internal pathology became vacant. By a vote of the council of the medical faculty, Hardy was elected to fill one of them. In this contest he was victorious over Charcot. Hardy retained this position until 1876, when, being chosen to succeed Bouillaud as professor of medicine, he took possession of his service at the Charité Hospital, where he remained until his retirement in 1887. Potain followed Hardy as professor of medicine.

At the time of Hardy's entrance into the field of dermatology, the classification of Willan and Bateman was generally recognized in France, due in a large measure to the influence of Biett who had brought it over from England. Hardy soon became imbued with the firm conviction that this classification was inadequate, and he did not hesitate to attack it boldly, even while conceding that it represented an important advance. Hardy believed that the subject should not rest there. He argued that limiting the study of a disorder solely to its external manifestations was not sufficient, since the skin lesions played

a secondary rôle, after all, and that cutaneous disorders should also be studied with reference to their true nature. He said:

> This research of the true nature of the skin affections has been totally neglected by the English school. . . . Founded on a study of the external characters, the system of classification of that school is, strictly speaking, only an artificial means of arriving at a diagnosis; but when thus solved, the diagnosis itself is incomplete for it reveals only a portion of the disorder, the external, leaving in darkness, the question of the etiology and the nature of the affair.

He divided the skin disorders into ten groups: (1) the macules and the deformities; (2) the local inflammations; (3) the parasitic dis-



ALFRED HARVEY, M.D.
1811-1893

orders; (4) the eruptive fevers; (5) the symptomatic eruptions; (6) the dartres comprising eczema, lichen, psoriasis and pityriasis; (7) the scrofulids; (8) the syphilids; (9) the cancers, and (10) the exotic disorders.

Hardy was among the first to embrace the ideas of Bazin relative to the parasitic skin disorders. He vigorously defended them as well as their author even at the Academy of Medicine where they had been assailed by Devergie. Bazin and Hardy became quite friendly, as is evidenced by their complimentary references to one another in their lectures.

The original description of the pigmentary syphilid and the quick cure for scabies were apparently the outstanding contributions of Hardy.

In a splendid obituary, Hallopeau credits him with describing the tuberculous gumma, which he spoke of as the phlegmonous scrofulid; scleroderma, which he termed sclérodermie oedémateuse, and mycosis fungoides, which he spoke of under the title of hypertrophic lichen. Hardy wrote extensively about lupus vulgaris, describing the acneic, the impetiginous and the papillomatous varieties. He placed dyshidrosis among the eczemas. Pelade or alopecia areata was contagious in his opinion. At an early date Hardy grouped herpes tonsurans, herpes circinatus and sycosis due to ringworm under the heading of cutaneous trichophytosis. He emphasized the arthropathy of psoriasis and also spoke of the bromide eruptions. It is told that Hardy often used rubber dressings in eczema, and he was a supporter of the use of mercurial preparations externally in acne vulgaris. Although a firm believer in adequate external therapy, he always stressed the importance of appropriate internal medication.

Hardy's textbook on general pathology was excellent, and several editions were printed. After succeeding to the clinic of Bouillaud, Hardy devoted himself largely to internal medicine. His articles on dermatologic subjects covered a wide range; he was also the author of no less than six works on dermatology. In 1858-1859, Hardy published a series of lectures on diseases of the skin. In 1862, his book on the datres appeared. Two years later, he wrote about scrofula and the scrofulids. The rest of his work comprises a treatise on diseases of the skin (1864), a photographic clinic of Hospital Saint Louis written in collaboration with Moysant and a practical and descriptive treatise of diseases of the skin (1886). Hallopeau refers to these works as follows, "All which he has written remains true, only the denominations have changed. His works can always be profitably consulted by those who come after him."

Brocq speaks of Hardy as follows:

> Alongside of Bazin, another physician at the Hospital Saint Louis, saw his clinics become very popular. Of great talent, clear, precise, simple in his descriptions, a direct disciple of Alibert, having had the good sense to abandon the cumbersome terminology of his teacher, to adopt that of Willan, of Plenck and of Rayer, but impregnated with a solid medical education and not disdainful of the general conditions of the body, such was the fortunate rival of Bazin, Professor Hardy. He had a marked hold upon the student body, as does always one who is clear, simple, not upset by difficulties and who has an easy and somewhat satirical elocution.

Hardy was an excellent teacher, being aided by a strong voice and clear speech. His manner was vivacious and authoritative, and his honesty and frankness were proverbial. He was most devoted to his friends and pupils. Parrot, J. Renaut, Landouzy, A. Robin, Dejerine, Hallopeau and Köbner of Berlin were among his most prominent pupils.

In his "Centenary of the Paris Medical Faculty," Corlieu refers to Hardy as an eminent clinician and one who was especially fond of physiology, but who was slow in adopting newer methods.

Hardy served as president of the Academy of Medicine. He was honorary president of the International Dermatological Congress which was held at Vienna in 1892. This was a striking honor both for him and for French dermatology.

Hardy died of pneumonia, Jan. 23, 1893, after only four days' illness. The phrase "Fuit vir probus et bonus, medendi et dicendi peritus" has been most appropriately applied to him. He may well be ranked among the leading dermatologists of France.

55 East Washington Street.

Correspondence

THE FIRST FRENCH EDITION OF FRACASTORIUS' SYPHILIDIS SIVE DE MORBO GALLICO LIBRI TRES: BY THE HONORABLE WILLIAM RENWICK RIDDELL

To the Editor.—In my edition of the "Poetical and Prose Works on Syphilis of Hieronymus Fracastorius," (Toronto, 1928), I said (p. 4) that the Syphilidis "does not seem to have found a French publisher until 1796, when Lucet issued a very beautiful edition (in 12mo.) accompanied by a translation into French and a Life of Fracastorius." I was misled by the little volume which I picked up on the left bank of the Seine on one of my visits to Paris, the title page of which reads: "SYPHILIS ou le mal vénérien, Poëme Latin de Jérome Fracastor, avec la traduction en François, et des notes.

Scilicet hâc tenui rerum sub imagine multum
Naturae, fatique subest, et grandis origo.

1796

A PARIS, Chez le Cen. Lucet, Directeur du Bulletin de de Littérature, rue Montmartre, No. 94."

This volume in the Avertisement, after lamenting that the work of Fracastorius was to be found in France only in the rich libraries, goes on to say that "the Syphilidis . . . has had many translations . . . in foreign countries but there is wanting one in our language and we have ventured to undertake the task of supplying one." I naturally supposed that this was the first publication, forgetting the fact that Lucet was a notorious thief of other people's literary goods.

I owe to the kindness of Dr. Montgomery of San Francisco, a volume with the title page: "Syphilis ou le mal vénérien, Poëme Latin de Jérome Fracastor, avec la translation en François, et des notes (A steel engraving representing the god, Apollo with his lyre holding a plaque on which is the portrait of Fracastorius, under which is a scroll with the inscription *'Deus haec quondam dignatus Apollo est.')*

A PARIS, Chez JACQUES-FRANÇOIS QUILLAU rue Saint Jacques, aux Armes de l'Université———M,D,CC,LIII, Avec Aprobation et Privilege du Roy." The portrait of Fracastorius on the plaque is reproduced in the Lucet edition on a separate page, opposite the title page.

I find that the Lucet edition is a copy of the former edition of 1753; there are, indeed, trifling differences, but these are either errors of the Lucet edition or misprints. For example, there is in Lucet, "quoque" for "quove"; "aeteris" for "aetheris"; "sagrae" for "Sagrae"; "Hypernus" for "hibernus"; "gallus," a cock for "Gallus," a Frenchman; "pupibus" for "puppibus," etc.; on the other hand, one finds in the early text "idones" for "idonea," and similar misprints.

The Lucet volume, which I took at its face value, is a plain literary steal—text, translation and notes. And I hereby apologize to Jacques-Francois Quillau, rue Saint Jacques.

Osgoode Hall.

WILLIAM RENWICK RIDDELL, LL.D., D.C.L., F.R.H.S., etc.
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Abstracts from Current Literature

Boecks' Sarcoid. Report of a Case with Visceral Involvement. Mitchell Bernstein, Frank Konzlemann and D. M. Sidlick, Arch. Int. Med. **44**:7 (Nov.) 1929.

A case of Boecks' sarcoid with visceral involvement is reported. Postmortem examination revealed lesions similar to those of the skin in the epicardium, bronchial mucosa and mucosa of the ileum. The authors feel that Boecks' sarcoid is a chronic infectious granuloma that may be caused by syphilis as well as by tuberculosis. In support of this, the Wassermann reaction of both the blood and the pleural fluid was strongly positive. The patient had severe dyspnea, which is not common in tuberculosis of the lung, but is a prominent symptom in syphilis of the lung. Examination of the sputum gave repeatedly a negative reaction for tubercle bacilli. Inoculation of the pleural fluid into guinea-pigs resulted in a negative reaction; and on section there were areas of perivascular and perineural mononuclear infiltration and independent collections of mononuclear cells in the liver and elsewhere.

It is pointed out that most studies of the etiology of Boecks' sarcoid are concerned with the presence of a concomitant tuberculosis, with the result that the presence of systemic lesions are often overlooked. Infiltration of the lungs, beginning at the roots and spreading toward the bases or upward, and associated with clear apexes is not common in tuberculosis, but is consistent with syphilis, and the authors feel that when patchy, discrete infiltrations of the lungs are associated with Boeck's sarcoid, they are sarcoid lesions. They have under observation another patient with sarcoid lesions. The history and the Wassermann reaction on the blood suggest syphilis, but the results of the examination of the chest suggest chronic tuberculosis. The authors were unable to isolate tubercle bacilli from the sputum.

Rattner, Chicago.

Etiology of Oroya Fever XV. Effect of Immune Serum on the Course of Bartonella Bacilliformis Infection in Macacus Rhesus. H. Noguchi, H. R. Muller, E. B. Tilden and J. R. Tyler, J. Exper. Med. **50**:355 (Sept. 1) 1929.

If convalescent serum was injected before inoculation with verruca peruana, the development in the blood of *Bartonella bacilliformis* was prevented in most instances, and the development of the skin lesions was delayed for considerable periods.

If the injection of the convalescent serum was made after the appearance of the skin lesions, the blood became free from *Bartonella bacilliformis,* but the nodules developed unchecked.

It is thought that a similar use of convalescent serum in man might be of value.

R. C. Jamieson.

Leprosy—An Unusual Case of Family Infection. E. A. Neff and R. J. Snodgrass, J. Trop. Med. **32**:137 (May 15) 1929.

The author reports his observations on a leprous family in which the parents and six children were all infected. One child escaped, not having come in contact with the first member affected.

Observations on Cryptococcus Castellani Re and Cryptococcus Gracioides Castellani. C. A. Mallardo, J. Trop. Med. **32**:145 (June 1) 1929.

A discussion of the cultural characteristics of these two organisms is made.

INVOLVEMENT OF THE HAIRY SCALP IN LEPROSY: A REPORT OF TWO CASES. E. A. NEFF and R. J. SNODGRASS, J. Trop. Med. **32**:147 (June 1) 1929.

Two cases are reported illustrating the rare involvement of the scalp in this disease. They were said to be the first reported in Makogai, Fiji Islands, since 1911.

TREATMENT OF EPIDERMOPHYTOSIS (DHOBIE ITCH) WITH CASTELLANI'S FUCHSIN PAINT. T. A. MAXWELL, J. Trop. Med. **32**:148 (June 1) 1929.

Good results are reported from the use of this paint — particularly in cases resistant to Whitfield's ointment and other standard applications.

REMARKS ON THE SO-CALLED "CULTURES OF DONOVAN BODIES." A. CASTELLANI and R. W. MENDELSON, J. Trop. Med. **32**:148 (June 1) 1929.

The question is raised as to whether the cultures are true cultures of Donovan bodies on account of not producing lesions of granuloma inguinale after inoculation, and because vaccines made from the cultures had no influence on the cases of granuloma inguinale in which they were used. If the cultures were true cultures of Donovan bodies, these bodies could not be considered the true causative agent in granuloma inguinale, in which case it is suggested that they would be "nosoparasites" — parasites constantly associated with the true causative organism.

DERMATITIS PAPULOSA NIGRA. A. CASTELLANI and C. W. DUVAL, J. Trop. Med. **32**:149 (June 1) 1929.

This disease was found to be extremely common in negroes of Jamaica and Central America — a few cases being found in Louisiana. The lesions often begin about the age of puberty and become progressively more marked as the age increases.

The etiology is unknown, race being considered a predisposing factor.

The lesions must be differentiated from juvenile verrucae, senile warts and moles. They appear to last the lifetime of the patient.

Treatment is unsatisfactory, cautery being required to destroy the growths.

R. C. JAMIESON.

INDUSTRIAL DERMATOSES: THEIR CAUSATION, RECOGNITION, PREVENTION AND TREATMENT. S. OVERTON, Brit. J. Dermat. **41**:255 (July) 1929.

Overton, in discussing industrial dermatoses, comments on the fact that returns from the Workmen's Compensation Act for 1927 show that for factories more certificates of disablement were issued for conditions of the skin than for any other compensatable condition. He stresses the fact that there has been an increasing number of claims on account of dermatitis contracted by the worker in the course of his employment. Industrial dermatitis may be produced by a vast and ever-increasing array of irritants of animal, vegetable or mineral origin. For the last three years, alkalis have held first place in the reports of the chief inspector of factories. Degreasing agents, such as turpentine and substitutes, paraffin, petrol and naphtha, sugar, dyes, particularly sulphur and basic, and "accelerators" that were used to shorten time in vulcanizing rubber were found to be among the important causes of industrial dermatitis. Engineers, bakers and flour confectionery workers, platers, dyers and calico printers, in the order named, constituted the groups in which the heaviest toll was levied. As a result of practical experience, the author has found the following suggestions to be of value in the prevention of industrial dermatoses: (1) the use of emollients before and after work; (2) complete removal of the irritants encountered at work, by harmless methods, several times per shift; (3) inspection of hands and arms by experienced observers to enforce (*a*) early treatment of minor injuries, cuts and burns, (*b*) scrupulous cleanliness of hands, arms and overalls, (*c*) removal from irritants and prompt treatment in early cases.

The importance of recognizing that a dermatosis is of industrial origin is discussed by Overton, and the fact that certain irritants produce localized lesions, besides causing a dermatitis, such as chrome and alkalis, is mentioned. The site of the dermatitis and knowledge of the irritants encountered in apparently innocuous occupations are among the subjects also briefly discussed.

INDUSTRIAL DERMATOSES: THEIR CAUSATION, RECOGNITION, PREVENTION AND TREATMENT. W. DYSON, Brit. J. Dermat. **41**:264 (July) 1929.

Dyson discusses those cases which are indistinguishable from cases of dermatitis commonly described as eczema. Such cases are commonly seen in bleachers, dye workers, chemical workers, plasterers, rubber workers, french-polishers and hair dressers. He recognizes no difference between an eczematous occupational dermatitis and eczema. Any condition which predisposes to eczema will also predispose to occupational dermatitis. Hyperhidrosis is a common factor in its production and xerodermatous and ichthyotic subjects are more susceptible than others. Idiosyncrasy is mentioned; and the author discusses the subject of sensitization and states that prolonged or repeated attacks of dermatitis may produce a general sensitization which has many of the characters of an "anaphylactic state." Seborrhea and secondary pyogenic infection, in his experience, favor the development of a general sensitization, although it may develop without the presence of any of these factors.

Reference is made to McGlasson's work on blood sugar content in dermatitis. Dyson examined fifty patients with all types of eczema and eczematous occupational dermatitis with this point in mind, and found that when pruritus was intense and there was no discharge, the average blood sugar content was high, but when the inflamed surface was discharging, the average blood sugar content was less. It was suggested that these results might be due to the liberation of histamine from the epithelium, which causes a liberation of glycogen from the liver by stimulating the secretion of epinephrine, and experiments are cited in which injections of histamine were given to raise the blood sugar content, with negative results. Experiments were also conducted by giving small repeated doses of the toxin producing the anaphylactic state, if sensitization is such a state, to determine whether an anti-anaphylactic state could be established and the patient could be gradually desensitized. One cubic centimeter of either blood serum or whole blood was injected at weekly intervals in seventeen cases. In ten of the cases, the patients made steady progress toward recovery; in most cases, the itching ceased after one or two injections. Only one of these patients has had a relapse to date. In one case the condition had cleared up after the patient had received eleven injections. A relapse within seven weeks followed, however, and the patient showed no improvement on subsequent injections. Four other patients showed no response to treatment, and in two cases the injections were discontinued.

A CASE OF METASTATIC SCIRRHOUS CARCINOMA OF THE SCALP. W. N. GOLDSMITH, Brit. J. Dermat. **41**:270 (July) 1929.

A case is described in a woman, aged 70, who presented lesions of an obscure character on the scalp of three months' duration which, on repeated histopathologic examination finally proved to be a metastatic scirrhous carcinoma, evidently blood-borne from the right breast, which had been removed six months previously. At the time of the examination, the patient presented an infiltration of the skin around the wound similar to that seen in the scalp, with lymphatic extension into the right axilla. Two photographs of the case and two photomicrographs illustrating the histopathology accompany the article.

WIEN, Chicago.

IONIZED SILVER IN THE TREATMENT OF BURNS. L. SHILLITO, Brit. M. J. **2**:668 (Oct. 12) 1929.

In a few cases, burns have been treated in the following manner, with good results. A solution of from 1 to 5 per cent silver nitrate is sprayed on the affected area, which is then exposed to ultraviolet ray.

The silver nitrate is ionized by the light and almost instantly there is a combination of silver ions with cell proteins. A protective coagulum is formed, as with the tannic acid method, but with the additional advantage of the sedative action of the ultraviolet rays on the nerve endings. No dressings are used. The treatment may be repeated after twenty-four hours.

RATTNER, Chicago.

BLASTOMYCOSIS. M. SYDNEY THOMSON, Proc. Roy. Soc. Med. **22**:1535 (Oct.) 1929.

A patient said that she had not suffered from skin trouble until three years before examination, when she had a dental abscess in the lower part of the right side of the jaw. This abscess burst through the skin and was succeeded by a swelling below the left eye. The latter lesion gradually softened and decreased in size until about eighteen months before examination, when a sinus appeared on the left side of the neck. Shortly a small "pimple" was noticed on the left side of the nose. This "pimple" had extended ever since in spite of all forms of treatment. When the patient was first seen, the submandibular gland was swollen and inflamed. There was an ulcer below the left lower lid the size of a threepenny piece, and it was heavily crusted over. The edge was clearly defined, and removal of the scab showed it to be an eighth of an inch in width and made up of closely packed papillomatous processes. Much pus was extruded from the center of the ulcer, leaving an ill defined hole the size of a hazelnut. Scattered over the face were four or five small scabs, which covered tiny papillomas and a pus area. The nose was grossly swollen and covered with large crusts. The anterior nares contained scabs, pus, and papillomas; the passage of the left nostril was entirely occluded. Administration of potassium iodide was begun five days before, and the dose had been gradually increased. Already there was definite improvement; the papillomatous processes were flatter and less distinct, and the patient herself volunteered the statement that she can breathe to some extent through the left nostril for the first time in two months.

VON RECKLINGHAUSEN'S DISEASE. A. M. H. GRAY, Proc. Roy. Soc. Med. **22**: 1536 (Oct.) 1929.

A boy, 15 years of age, who apparently had had an eruption ever since he was a baby, showed many neurofibromas; the median and ulnar nerves on both sides were enormously enlarged, and showed nodules all the way down. The median nerve could be traced to the wrist, and the ulnar nerve almost equally well. Curiously, the boy had no symptoms, in spite of this nerve involvement, and he had no pain unless definite pressure was exerted on the nerves. He had the three classic features of the disease: molluscous tumors (not large), neurofibromas on the nerve-trunks and patches of pigmentation on the trunk, neck and back. There was no history of any similar condition in the family, so far as could be ascertained. The boy's mental condition was normal.

ACQUIRED EPIDERMOLYSIS BULLOSA (?). G. B. DOWLING, Proc. Roy. Soc. Med. **22**:1536 (Oct.) 1929.

The patient, an 18 year old boy, three years ago had blisters which began to develop, chiefly about the ankles, but later they appeared on other parts of the body. Each lesion began as a small bulla, that enlarged, broke down and frequently became secondarily infected and ulcerated. The lesions healed and left scars. They gave rise to no irritation, but were rather painful when they broke down. They were found chiefly about the knees, ankles, extensor aspects of the legs, hands, extensor aspects of arms and forearms, elbows, buttocks, and there were a few on the ears. The thighs, abdomen, and back were free. There was no family history of epidermolysis bullosa. The patient was healthy in other respects. Since the case was shown, the question of artefact has been tested. A soft zinc paste had been applied to the legs for two successive weeks; after

the first week, several fresh bullae were found beneath the dressing. The observation was repeated with the same result. It was noted that the dressing had not been tampered with in any way.

GUY, Pittsburgh.

STERILITY IN PATIENT WITH TABOPARESIS CURED BY MALARIA THERAPY. M. WEISSMANN, Wien. klin. Wchnschr. **42**:878 (June 27) 1929.

Weissmann reports the case of a woman, aged 32, who had been married for sixteen years. In spite of the fact that she never used contraceptives, she did not become pregnant. Examination revealed typical taboparesis. The joints were swollen and very painful. She suffered from severe headaches and was completely deranged. The Wassermann reaction was positive in the blood as well as in the cerebrospinal fluid. The patient was given malaria treatment and after that her condition improved markedly. The speech and mental disturbances disappeared. Ten months after the completion of the malaria treatment she gave birth to a healthy child. After that the pains in the joints recurred with greater severity. The psychic disturbances, however, did not recur. Two years later she had a second confinement, and the year following a pregnancy in the fifth month was interrupted. Since the malaria treatment four years previously, the psychic disturbances had not recurred. At present she does not have signs of taboparesis. Serologic tests of the cerebrospinal fluid always give negative reactions. Her general condition likewise is improved. The author thinks that the functioning of the whole organism was changed by the malaria treatment.

INDICATIONS AND CONTRAINDICATIONS FOR TREATMENT OF LATENT SYPHILIS. O. NAEGELI, Schweiz. med. Wchnschr. **59**:697 (July 6) 1929.

Many authorities are of the opinion that antisyphilitic treatment is not advisable in cases of latent syphilis. After discussing the various factors that are cited by these authors as indications against antisyphilitic treatment, Naegeli comes to the following conclusions: In patients who have never been treated or in whom the treatment has been discontinued for a long period, therapy is advisable. If such patients do not receive proper treatment the disease may involve internal organs that so far have not been affected, or skin eruptions may occur. It is also pointed out that during these latent periods the chances for a cure are better. In instances in which a definite time of infection cannot be determined, treatment is likewise advisable. In pregnant women with syphilis, antisyphilitic treatment should be instituted immediately in order to protect the fetus. Infants, children and adults with congenital latent syphilis should also be given antisyphilitic treatment. Contraindications to antisyphilitic therapy are: serious intercurrent diseases and idiosyncrasy to antisyphilitic medicaments. In patients who are over 70 years of age, a positive serologic reaction should not be considered an indication for antisyphilitic therapy, nor should antisyphilitic treatment be resorted to in patients who have undergone intensive treatment several times and in whom a positive serologic reaction is the only indication of a syphilitic infection.

DERMATOLOGIC ABSTRACTS

THE JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION

MYCOTIC VULVOVAGINITIS. N. W. POPOFF, F. FORD and W. H. CADMUS, Am. J. Obst. & Gynec. **18**:315 (Sept.) 1929.

Popoff et al. report a case of mycotic vulvovaginitis encountered in a woman, aged 25, who first noticed the clinical symptoms of the disease at the beginning of the sixth month of pregnancy. The chief complaint at this time was the marked

pruritus of the vulva and vagina, a burning sensation in the vagina, and some discharge. The symptoms were persistent and progressively disturbing. The cause was found to be *Monilia psilosis.*

THROMBO-ANGIITIS OBLITERANS. M. E. BARRON and H. LINENTHAL, Arch. Surg. **19**:735 (Oct.) 1929.

Analysis of thirty-four cases made by Barron and Linenthal demonstrated that this disease is not confined to vessels of the extremities, as is generally accepted, but is a generalized disease that may affect any part of the arterial tree, including the coronary arteries, the abdominal arteries and the arteries of the brain.

FINAL RESPONSE OF SMALL CUTANEOUS VESSELS. P. ROUS and H. P. GILDING, J. Exper. Med. **50**:489 (Oct. 1) 1929.

A study made by Rous and Gilding of the blanchings (Bier's spots) which develop in human skin deprived of circulation has proved them referable to the same general causes that lead to ischemic patching of the skin of animals, and that they are conditioned by the same factors. Both are expressions of a secondary contraction of vessels that have become hyperirritable owing to an inadequate circulation. The contraction is favored by emptying the vessels, but it occurs pronouncedly even when there is a trickle through them of arterial blood. In the case of man the contractile impulse is sometimes so great as to overcome the maximum pressure that the vessels will support.

TREATMENT OF ECZEMA IN INFANTS BY A MILK-FREE REPLACEMENT DIET. H. M. ROBINSON, South. M. J. **22**:936 (Oct.) 1929.

Robinson has prepared a routine diet list for these cases: lamb and beef broth cooked with barley or rice, the broth to be thickened with flour, and croutons used always. Cereals include corn meal, Quaker oats, cream of wheat or farina, rice, barley, hominy. All cereals are to be cooked three hours and put through a sieve. Vegetable purées include tomatoes, spinach, carrots, peas and lima beans (fresh or dried). Grain includes potatoes (white or sweat, baked or mashed); string beans, macaroni (put through a sieve), spaghetti, soy bean flour, wheat bread, and crackers (not sweet). Also coddled white of egg is used. After the age of 8 months, children are given shredded or ground meats: beef, lamb or chicken. The following three articles are mentioned primarily to condemn because almost all children have reacted against these: orange juice, prune juice and apple sauce. Cod liver oil is given, ½ drachm (15 cc.), three times a day, to be increased slowly. The age of the child determines the quantity of feeding.

TREATMENT OF TINEA CAPITIS: COMPARISON BETWEEN ROENTGEN RAY AND THALLIUM. J. L. PIPKIN, Texas State J. Med. **25**:335 (Sept.) 1929.

Pipkin relates his experience with thallium and the roentgen ray in the treatment of tinea capitis. Twenty-seven cases were treated with protoxide of thallium acetate. The Buschke technic was followed. Only one case in the group showed a recurrence of the disease. There were no cases in which the hair failed to fall, although in four cases manual epilation had to be resorted to in order to get complete baldness. Only seven patients were entirely free from toxic manifestations. Two patients showed severe toxic symptoms. Thirty patients were treated by complete epilation with roentgen ray according to the MacKee and Remer modification of the Adamson-Keinbach method. The period of baldness lasts from three weeks to two months, This fact being a decided advantage over the thallium method of epilation, as the danger of reinfection of the hair is eliminated. Regrowth starts in from one to three months after epilation, which allows ample time for local treatment. An interesting feature is a change in the quality of the hair. At times there is a change in color or texture. Again, children with straight hair may get a regrowth of curly hair or vice versa. The advantages of each of

these methods of epilation are discussed. The use of the thallium method requires a knowledge of the routine of a very simple procedure, necessitating no skill and but an average amount of intelligence and precaution on the part of the administrator. The only preliminary preparation for thallium medication is the urine examination and careful weighing of the child. The method of epilating by means of the roentgen ray requires a skilled, experienced operator who has had the necessary roentgenologic training. Because of this, and also because epilation carries with it a responsibility and is tedious, the operation becomes expensive to patients. Although thallium can be given to very young children with no trouble as regards administration and with apparently the best results, it should not be used for patients past 12 years of age, or after any of the signs of puberty have appeared. There are some cases in which thallium is contraindicated, for example, any case complicated by nephritis or any other organic disease. The roentgen ray cannot be used in patients who have been recently treated (within two or three weeks) with irritative local applications, or who have had recent fractional doses of the roentgen ray. The roentgen ray can be used with impunity in adults. In this series it seemed that underweight children tolerated thallium better than those of normal weight. But in cases in which either method is available, the possible dangers associated with one or the other should be considered in the final choice of a method to eradicate the disease. Thallium causes falling of the hair by its action on the sympathetic nervous system. It also has some influence on the endocrine system. The drug is eliminated mostly through the kidneys. No one knows what remote effect it will have on the kidneys, endocrine glands, and the sympathetic nervous system. It does not seem feasible to use a drug as toxic, and with such uncertain possibilities as thallium acetate, in the treatment of ringworm of the scalp, if the harmless, established, safe method with the roentgen ray is available.

PATHOLOGY IN THE TROPICS. R. M. CHOISSER, U. S. Nav. M. Bull. **27**:551 (July-Oct.) 1929.

Five cases of basal cell tumors and three of nonpigmented melanomas were found in pure-blooded Haitians. The former were on the face and scalp and the latter on the heel. The pathologic changes of late untreated yaws so closely resemble those resulting from tertiary syphilis that it was impossible to differentiate the two without a clinical history. The first pathologic evidence of yaws infection in the internal organs was found in the aorta. These early lesions manifest themselves by irregular linear scars and atheromatous plaques, which begin about 5 mm. above the aortic cusps. The aortic valves themselves do not appear to be universally involved in tertiary yaws or in their late cases of so-called trepanematosis. Late untreated yaws predisposes to aortic aneurysm, particularly of the arch.

EARLY STAGES OF ORAL CANCER. G. T. MOWAT, Glasgow M. J. **31**:121 (Sept.) 1929.

In a series of 244 cases of oral cancer analyzed by Mowat, the large majority showed a long period of chronic irritation of the part affected, which could have been avoided and the onset of the epithelioma probably averted by removal of the irritant at an early stage.

ERYSIPELAS: EXPERIMENTAL STUDY OF BIOLOGIC TREATMENT. V. DE LAVERGNE, P. FLORENTIN and H. GOUSSET, Paris med. **2**:180 (Aug. 31) 1929.

De Lavergne et al. report that in their experiments on rabbits, erysipelas proved obdurate to antistreptococcus vaccine or serum therapy. Neither the local application of ointments containing antistreptococcus vaccine nor the subcutaneous injection of vaccine or serum into patients and animals had any influence on the duration or severity of the disease.

PUERPERAL SCARLET FEVER. P. BAIZE and M. MAYER, Presse méd. **37**:1007 (Aug. 3) 1929.

On the basis of a personal study of six cases and a review of the medical literature on the subject, Baize and Mayer state that in the puerperium two forms of true scarlet fever may be observed, namely, the form that is the result of an intercurrent infection and the form that is the result of an endometritis. True scarlet fever should not be confused with the scarlatiniform puerperal erythemas from which it can be differentiated by biologic tests, particularly the Schulz-Charlton reaction.

UNILATERAL SYNDROME RESEMBLING THAT OF RAYNAUD'S DISEASE AND DUE TO SUPERNUMERARY RIB. P. CAPONE-BRAGA, Policlinico (sez. prat.) **36**:1281 (Sept. 9) 1929.

Capone-Braga recites a case of cervical rib associated with vasomotor phenomena resembling those occurring in Raynaud's disease. The case is of interest for two reasons: the post-traumatic beginning of the syndrome and the marked improvement following the removal of the supernumerary rib. The question that arose was why the supernumerary rib during the course of the many years it existed attracted no attention to itself, but did so immediately after a trauma called forth a series of disturbances.

ROENTGEN THERAPY OF PLANTAR WARTS. M. MIRANDA GALLINO, Semana méd. **36**:689 (Sept. 5) 1929.

Ten patients, aged from 22 to 54, with plantar warts, treated with roentgen rays by Miranda Gallino, made a complete recovery. The author says that his series is too small to justify the conclusion that all cases of plantar warts should be cured by this treatment, but his good results support the optimistic opinions concerning this treatment. All patients but one received one or two treatments; in one patient three treatments were necessary. Roentgen irradiation, when judiciously used, is harmless and it should be the treatment of choice in plantar warts.

TREATMENT OF DERMAL LEISHMANIOSIS. A. DOSTROWSKY, Arch. f. Schiffs-u. Tropen-Hyg. **33**:417 (Aug.) 1929.

On the basis of a comparative study of his results in the treatment of nine patients with dermal leishmaniosis with arsphenamine, nineteen with antimony and potassium tartrate, ten with carbon dioxide snow and thirty with roentgen irradiations, Dostrowsky states that roentgen treatment gives the best results. The advantages of roentgen treatment are that it requires a relatively short time and is painless. The disadvantages are that it is expensive and necessitates the presence of a roentgen laboratory. The author believes that roentgen irradiations should not be used in the treatment of dermal leishmaniosis in small children because the penetrating rays might injure growing organs under the skin. In cases in which roentgen irradiations cannot be used, treatment with carbon dioxide snow should be considered.

EFFECT OF FRESH SYPHILITIC INFECTION ON CLASSIC ATTACKS IN TERTIAN MALARIA. G. J. PEREKROPOW, Arch. f. Schiffs-u. Tropen-Hyg. **33**:432 (Aug.) 1929.

Perekropow reports the case of a girl, aged 19, in whom the appearance of a severe secondary syphilid was followed by the disappearance of typical attacks of tertian malaria. The patient was not undergoing treatment for malaria at the time the attacks disappeared; in spite of the fact that examinations of the patient's blood three weeks before the syphilid appeared had revealed great numbers of tertian parasites, repeated examination of the blood after the syphilid appeared did not reveal the presence of a single malarial parasite. On the basis of this

case and two similar cases the author believes that the good results obtained in the malaria treatment of paresis are to be attributed not to the action of the malarial parasites but to the combined treatment with arsphenamine and quinine.

CHYLOUS CYSTS OF NECK. J. VOLKMANN, Beitr. z. klin. Chir. **146**:654 (June 12) 1929.

Volkmann reports the occurrence of this condition in three patients. The first, aged 34, six months previously had developed a swelling on the right side of the neck which rapidly increased and was diagnosed as a cystic lymphangioma. On puncture, however, 28 cc. of a cloudy yellowish fluid exuded and a diagnosis of chylous cyst was made; chemical examination confirmed the diagnosis. The cyst was removed; in the course of the operation it ruptured and chyle flooded the wound. The patient recovered, however, within a short period. A slight swelling recurred a week later but it receded and has not reappeared. The second case concerned a woman, aged 23, in the seventh month of pregnancy. Two years previously she had developed a swelling on the right side of her neck which on puncture yielded pus. The author withdrew 120 cc. of fluid from the cyst and injected 5 cc. of iodoform in oil. The swelling receded entirely. Two years later the patient returned with a recurrence of the swelling. A culture of the fluid withdrawn remained sterile; the chemical composition of the fluid was similar to that from the first patient. The cyst filled again one month later and 46 cc. of fluid exuded on puncture; this time 5 cc. of iodized sesame oil 20 per cent was injected. The cyst filled again after six months, the patient still refusing an operation. Sixty-nine cubic centimeters of fluid of the same composition was withdrawn from the cyst. In a third patient, aged 24, a swelling on the left side of the neck had persisted for two years. Roentgen and ultraviolet irradiations were used on the assumption that it was of tuberculous origin. As no effect was evident after eighteen irradiations, the tumor was punctured. A milky white fluid exuded, conforming in chemical composition to the fluid found in the previous cases. Thirty cubic centimeters of fluid was withdrawn. One month later, two small nodules were still felt; after three months only one was noted at the site of the cyst. Volkmann reviews the literature on the subject and discusses the etiology of the condition.

DIETARY TREATMENT OF LUPUS VULGARIS. S. BOMMER and L. BERNHARDT, Deutsche med. Wchnschr. **55**:1298 (Aug. 2) 1929.

Bommer and Bernhardt point out that the older methods of treatment of lupus vulgaris usually aimed at the destruction of the focus of the disease. However, this local therapy often resulted in disfiguring scars. For this reason it has been more and more abandoned in favor of a general therapy. The main feature of this treatment is the diet, which is based on the rules given by Gerson, Sauerbruch and Herrmansdorfer. This dietary treatment completely eliminates the use of salt in the preparation of the foods. Fats are given in comparatively large quantities; carbohydrates, however, are restricted. A general irradiation, in the form of sun baths, often adds to the favorable results of the diet. However, local irradiation of the diseased areas is not advisable. Under the influence of this treatment, patients with severe lupus vulgaris gradually recover; the author shows pictures that illustrate the good cosmetic results of this therapy.

CLINICAL OBSERVATIONS IN ENCEPHALITIS FOLLOWING SMALLPOX VACCINATION. H. G. HUBER, Deutsche med. Wchnschr. **55**:1370 (Aug. 16) 1929.

Huber reports the cases of three children, aged between 1 and 2 years, in whom disturbances of the central nervous system developed after smallpox vaccination. Two of these patients showed the typical symptoms of encephalitis. In one of them the symptoms developed on the fifth day after the vaccination, and the vaccine virus could be demonstrated in the spinal fluid. In the second case, a flaccid paralysis of the right arm remained after the other symptoms had dis-

appeared. In the third patient the nervous manifestations became noticeable on the twenty-fourth day after the vaccination. However, because catarrhal disturbances intervened between vaccination and the appearance of the nervous symptoms, it is not certain whether the vaccination was the cause of the postvaccinal encephalitis. It is pointed out that opinions differ as to the duration of the incubation in this disease.

SIMULTANEOUS OCCURRENCE OF CONGENITAL AND ACQUIRED SYPHILIS IN INFANTS. E. HOFFMANN, Deutsche med. Wchnschr. **55**:1289 (Aug. 2) 1929.

According to Hoffmann, clinical as well as experimental observations prove that in infants congenital and acquired syphilis may occur simultaneously. An infant who at birth appears healthy but who shortly before birth has been infected by way of the placenta may acquire a superinfection in the form of a cutaneous chancre. The chancre is accompanied by enlargement of the regional lymph nodes and often simulates the primary lesion in acquired syphilis. Whether the spirochetes that enter the organism during the placental infection, or whether the spirochetes that cause the cutaneous infection, will dominate depends on the number of spirochetes that caused the superinfection. The author asserts that the mixed forms of syphilis are comparatively rare. The differential diagnosis in such cases is usually difficult. Nevertheless it is not only of theoretical interest but also of practical value, and it requires the cooperation of gynecologists, dermatologists and pediatricians.

USEFULNESS OF SEVERAL NEW TURBIDITY AND FLOCCULATION REACTIONS FOR SEROLOGIC DIAGNOSIS OF SYPHILIS. H. GROSS and L. OELRICHS, Klin. Wchnschr. **8**:1537 (Aug. 13) 1929.

Gross and Oelrichs performed tests to determine the relative value of the serologic reactions used in the diagnosis of syphilis. The Wassermann test is still the standard method. However, there are several turbidity and flocculation reactions that may be performed to supplement and to control the Wassermann test. The authors examined 1,500 serums. In addition to the Wassermann test they employed the following tests: Kahn's, Bruck's, Dold's, Meinicke clarification, Meinicke turbidity and Sachs-Witebsky's citochol (*München. med. Wchnschr.* **76**:657 [April 19] 1929; abstr., *J. A. M. A.*, July 6, p. 82). They come to the conclusion that besides the Wassermann test several turbidity and flocculation tests should always be done. It was found that the Kahn test, the citochol reaction and the Meinicke turbidity reaction were the most useful supplementary tests, not only because they are relatively simple and rapid but also because in regard to their sensibility and their specificity they are equal or even superior to the Wassermann test.

TREATMENT OF CARBUNCLE OF NECK. O. POLLWEIN, München. med. Wchnschr. **76**:1377 (Aug. 16) 1929.

Pollwein states that patients with carbuncles should be treated in a hospital rather than in the physician's office or in the home, because in the hospital there are all the facilities that make a proper treatment possible. The author then describes a technic which has given satisfactory results. The carbuncle is completely laid open by a horizontal incision that goes parallel with the natural lines in the skin. Additional incisions are made vertical to the first incision. This method is said to be superior to the crucial or starlike incisions, because it does away with the narrow, pointed skin flaps that usually become necrotic; it gives better cosmetic results, and it also shortens the period of treatment. To make the operation painless, it is performed under ethyl chloride narcosis. A local anesthetic is not sufficient. After proper surgical treatment frequent changing of the dressing will not be necessary. The first tampon may remain about ten days. For the after-treatment the author recommends suture of the incision. Eight days later the wound is healed enough so that further treatment will not be necessary.

METASYPHILIS OF CENTRAL NERVOUS SYSTEM: EARLY RECOGNITION AND PREVENTION. E. HOFFMANN and A. M. MEMMESHEIMER, Nervenarzt. **2**:399 (July 15) 1929.

Hoffmann and Memmesheimer stress the importance of adequate treatment in early cases of syphilis to prevent later manifestations in the central nervous system. They consider the Bonn method of treatment, in which arsphenamine and bismuth compounds are given in large frequent doses, as effective in recent cases of infection as well as in older cases. As a basis for this statement, the results in 1,000 patients from the records of the Bonn clinic have been made use of, and in addition the results in sixty cases from the private practice of Hoffmann and 115 cases from that of Memmesheimer are reported. When maximal chemotherapy is combined with malarial treatment the results may be still more favorable and metasyphilis may become a curable disease. The complete suppression of syphilis still remains problematic; congenital syphilis, however, can certainly be prevented.

LETHARGY FOLLOWING ROENTGEN IRRADIATION OF SCALP. A. DRUCKMANN, Strahlentherapie **33**:382 (July 23) 1929.

Druckmann reports the development of lethargy in thirty out of 1,100 children in whom, to shorten the irradiation time for epilation in ringworm, roentgen irradiations of a much higher voltage than is ordinarily prescribed were used. The cerebral reaction appeared from six to eight weeks after the irradiation. The children became apathic, did not talk and slowly whispered answers to questions. The parents stated that the children wanted to sleep continuously, were awakened with difficulty and ate little. They frequently went to sleep while sitting or even while standing. Some of them complained of headache and in some of them a temperature as high as 38.5 C. (101.3 F.) was noted. Changes were not found in the urine and blood; in a few cases the pressure of the cerebrospinal fluid was increased. The condition lasted from four to six days in light cases and fourteen days in severe cases, and then disappeared completely. The children have been followed four years; in none of them have late physical or mental injuries appeared.

TREATMENT OF MELANOSARCOMA. A. MÜLLEDER, Wien. klin. Wchnschr. **42**: 1019 (July 25) 1929.

Mülleder reports the case of a physician, aged 66. Since childhood the patient had had a pigmented mole on the left thigh. At the age of 56 he first noted an ulceration of the mole. In 1921 it was extirpated and the histologic examination revealed a melanosarcoma. In 1924 a growth about the size of a nut was removed from the groin on the left side. This tumor likewise proved to be a melanosarcoma. Several months later, other growths developed in the left inguinal region. The patient refused operation. However, in 1927 the tumors had enlarged and the patient consented to have them removed. In the meantime smaller growths had developed in the right inguinal region. These were removed in the fall of 1928. In each case, histologic examination confirmed the diagnosis: melanosarcoma. At the present time, small tumorous growths are developing again in the inguinal regions. Roentgen examination of all organs and of the bones revealed that the patient is otherwise free from metastases. Extirpation and histologic examination of one of the many nevus-like formations on the skin of the patient proved that they were not of a malignant nature. Although the patient has been suffering from melanosarcoma for eight years, his general condition is comparatively good. The author thinks that this is largely due to the patient's self treatment. He changed his mode of life completely. He abstained from meat and lived mostly on a raw vegetable diet. He also abstained from alcohol and from nicotine. The most significant factor in his treatment was a sun bath, which he took daily for several hours. In order to be able to continue his light therapy all the time, he spent the winters in a southern climate, and on cloudy days he took quartz lamp irradiations lasting from one to one and one-half hours.

Society Transactions

CHICAGO DERMATOLOGICAL SOCIETY

Regular Meeting, April 17, 1929

H. R. FOERSTER, M.D., *President, Presiding*

A CASE FOR DIAGNOSIS. Presented by DR. NOMLAND by invitation.

A healthy woman, aged 25, was first seen five weeks before presentation. At that time she complained of an eruption of two months' duration, which began as a small red area near her nose and spread outward and downward as a red elevated line. At either side of her upper lip were red elevated lines similar to the ones now present. A lesion appeared on her chin four weeks ago as a red slightly elevated macule.

The patient presented a round lesion about 5 cm. in diameter located beneath the chin. The center was clear; the periphery consisted of a red line about 2 mm. in diameter and was slightly elevated. There was no scaling. The patient's Wassermann reaction was negative. Five grains of phenolphthalein were given a week before presentation, but there was no exacerbation of the eruption the following day. Search for fungi had been made several times, with negative results. While she complained of frequent sore throat and mucous discharge from the nose, a thorough examination of the nose and throat was essentially negative.

DISCUSSION

DR. MITCHELL: I have never seen such a case, but the one presented here resembles the cases of erytheme annulaire centrifuge described by Darier and other French writers.

DR. ZEISLER: I had a similar impression, but I also thought of the possibility of an annular type of erythematous lupus. I have seen annular lesions in erythematous lupus several times where the spread was as rapid as in this case.

DR. ORMSBY: I think that is all that can be said. The patient was presented to demonstrate that the condition is not syphilis or an eruption due to phenolphthalein. In the early days some of the so-called cases of erythema perstans were eruptions due to phenolphthalein, but this case merely presents a persistent erythematous lesion of unknown etiology.

DR. NOMLAND: I had a similar case in a colored woman who had the lesion on the back of the neck. Biopsy showed an immense amount of subpapillary infiltrate of round cells, but the interesting thing was that the annular lesions disappeared within a week following the examination, presumably from the irritation of the iodine that was applied.

A CASE FOR DIAGNOSIS (PEMPHIGUS?). Presented by DR. SENEAR and DR. WIEN.

E. P., a man, aged 45, had noted an eruption on the tongue, lips, cheeks and face for three months, associated with only a mild subjective sensation. He said that about two years before presentation he had an ulcer in his throat. He had been using various gargles and mouth washes. He also said that the severity of the lesions varied from week to week, and that one month ago the mouth was practically free from eruption.

There was a marked fetor oris and the patient presented numerous ulcerations of a superficial character on the soft palate, the tongue, buccal mucosa and lips. There were numerous crusted lesions about the nose and face and a large broken

bulla could be seen at the umbilicus, which the patient said has been present for one month previous to examination.

DISCUSSION

DR. PARKHURST: From the appearance and history I think that this is a case of pemphigus.

DR. SENEAR: We regarded it as pemphigus, but thought it sufficiently interesting to present in view of the extensive lesions in the mouth. Cultures have been started but the patient was seen only two days before presentation and they are not completed.

DR. ORMSBY: I wish to call attention to the fact that the lesions in the mouth are different in this case than in the majority of such cases, in that this patient said that the lesions cause no discomfort. It is unusual to have no sensation from so many lesions in the mouth in pemphigus.

HODGKIN'S DISEASE. Presented by DR. WAUGH.

A man, aged 35, whose general health was good until the present trouble began four months before presentation, developed a papulopustular eruption on the thorax, shoulders, arms and neck. The lesions were numerous, bluish red, varying in size from that of a pinhead to that of a pea, and were rather deeply seated. They were not grouped and did not itch. New lesions appeared from time to time, and some atrophic scarring was observed. The patient was taking no medicine when the rash appeared. There was no history of any venereal disorder, and the Wassermann reaction of the blood was negative.

Coincident with the appearance of the rash, the cervical axillary and inguinal glands became enlarged, those in the left axilla forming a mass larger than a hen's egg. The blood showed no increase in the leukocytes, and a differential count showed a normal percentage.

The patient had lost 13 pounds (5.9 Kg.) in weight during the past four months. He complained of night sweats and at times a mild cough. Physical examination of the chest revealed nothing of importance.

DISCUSSION

DR. ORMSBY: I saw this patient on April 6, and at that time concluded that he had lymphogranulomatosis. The only way to demonstrate this would be to do a biopsy, but the patient and his physician objected. With the general adenopathy, I think that the condition might be considered a nonsyphilitic eruption in a case of lymphogranulomatosis.

SCROFULODERMA AND TUBERCULIDS. Presented by DR. SENEAR and DR. WIEN.

H. T., a girl, aged 11, was first seen in January, 1927, with a scrofuloderma of the left knee and region of the ankle and lesions of erythema induratum on the right calf. She subsequently developed lesions of folliclis and acne scrofulosorum on the trunk and extremities. She was given tonic quartz light exposures with a marked improvement in the condition of her skin and in her general condition. In January, 1928, fresh lesions of folliclis were noted on the trunk, and in November, 1928, a scrofuloderma developed in the region of the left lower jaw.

On presentation, the patient exhibited lesions of scrofuloderma of the jaw and similar involuted lesions at the left knee and ankle, erythema induratum of the right leg and lesions of folliclis on the trunk and extremities.

The Pirquet test was positive. Examination of the chest was negative for tuberculosis, and roentgen examination revealed no unusual amount of lung markings.

DISCUSSION

DR. OLIVER: I have seen pictures of this patient and thought that the lesions were a good example of scrofuloderma on the neck. The lesions on the legs, I think, are those of erythema induratum.

DR. ORMSBY: Years ago, in trying to determine the etiology of tuberculids, I collected a number of cases in which two or three definite tuberculous conditions were present together with the tuberculid, but I have never seen a case with four such conditions present. On account of the value of gold sodium thiosulphate in lupus erythematosus, which is in some instances related to tuberculosis, I have used it in three cases of erythema induratum and the lesions cleared up more rapidly than with other methods. Incidentally, I have treated subcutaneous sarcoid in the same way and it has cleared that up better than with arsenic, which was formerly used. These results are interesting as they seem to place the whole group in the same etiologic category.

DR. MITCHELL: I have treated one patient, with the erythema induratum type of lesions, with a gold preparation to the point where the patient did not wish to have any more, without any perceptible effect.

SPOROTRICHOSIS. Presented by DR. WHITE.

A German watchman, aged 65, said that "pimples" had appeared on his left wrist three years previous to presentation. Other "pimples" soon appeared and a line of such lumps extended up his arm, causing considerable pain. He was treated by the application of iodine externally and given some medicine by mouth, and was well in a short time. Five months before presentation the trouble recurred and extended in the same way, causing pain in the large nodules and a burning sensation in the left palm. He had pains in the knees and other joints for five years previous to presentation.

A group of nodules, brown, red and hard were seen on the left wrist, and, extending up the forearm and arm, was a series of such nodules connected by hard cords of infiltration. In all there were about twenty nodules, a few of which had softened and in these the organisms (sporotrichotic conidia) were found. There was no evidence of any inoculation lesion. The patient had received a small amount of treatment but had shown no improvement. Cultures were negative. One of the hard nodules was excised, and showed a dense infiltrate largely of large round cells, many of them showing mitotic figures, much resembling a sarcoma.

DISCUSSION

DR. ZEISLER: This case was very interesting on account of the primary focus, and because of the unusual sclerosis along the tract of the lesions. I consider it a typical sporotrichosis.

DR. FINNERUD: Histologically, to me the condition resembled a true tumor more than a granuloma. If such, it would fall in the sarcoma group. It was not my conception of the microscopic picture of sporotrichosis. This, of course, does not appear logical in view of the laboratory observations of Dr. Lawless.

DR. STILLIANS: No sporothrix could be cultured because the man was taking iodides when he came to us. I do not know whether it was made clear that he had similar attacks a number of years ago, which promptly cleared up under the administration of iodides. He was not under our observation at that time.

DR. WHITE: The microscopic recovery of the sporotrichotic organism from the pus is quite easy if Dr. Lawless' pioneer work is followed. They grow within from one to three hours and can be seen to do so under the microscope. So far as the biopsy is concerned, the tumor appeared to be a sarcoma, but I think the recovery of the organism proves the diagnosis, unless two different disease processes are present. The patient is a watchman and it is possible that he became infected from fruit crates, or something of that nature. No primary inoculation lesion has ever been present while under observation.

DR. FOERSTER: I believe that the pronounced fibrosis and lack of vascularity in the lesions may have been a factor in the failure of iodide medication.

A Case for Diagnosis (Lichen Nitidus?). Presented by Dr. Senear.

A boy, aged 8, was seen for the first time the day before presentation. He presented an eruption consisting of pinhead-sized shiny papules, some almost the color of the skin and some slightly brownish, scattered over the trunk. There had been a gradual increase in the extent of the eruption, and with the exception of a few lesions on the genitalia, none of them had disappeared. The boy was in good health, and there was nothing of interest in the general history. The condition was said to have been present one year. There was no subjective symptom.

DISCUSSION

Dr. Stillians: I am not an authority on lichen nitidus, but would offer that diagnosis.

Dr. Zeisler: I thought that the condition was lichen scrofulosorum.

Dr. Finnerud: I thought that it was an atypical lichen scrofulosorum. The lesions were not of classic shape or distribution, but otherwise they seemed to fit in well with that disorder.

Dr. Senear: I saw the boy with Dr. Oliver two days ago and we mentioned lichen nitidus and lichen scrofulosorum at that time. The diagnosis of lichen planus had been suggested previously, but we did not feel that it could be that disorder. As between lichen scrofulosorum and lichen nitidus, I felt that it was the later, in view of the lack of scaling and because the lesions were not shiny and many of them about the color of the normal skin. I took occasion yesterday to do some reading on lichen nitidus and I could see no point at which this case was at variance with the description. I feel that it is a generalized lichen nitidus. The lesions have disappeared from the penis.

A Case for Diagnosis. Presented by Dr. Senear and Dr. Wien.

C. W., a negress, aged 29, first noted an eruption on the face about eight years before presentation in the region of the right ear, and on the right forearm, that had gradually spread during the interim. For the past seven years the patient had been taking drops for a blood tonic at intervals. Ten years ago she received "shots" in the arm for a sore throat. She presented herself with a prescription containing a solution of potassium arsenite and a saturated solution of potassium iodide given her by her druggist. This particular prescription she had been taking for one week prior to their seeing her, and she said that the other drops taken by her were of similar character.

She now had a diffuse slate-like pigmentation in the region of both ears with extension down the neck and to the cheeks, and a scattered, diffuse, brownish reticular, pigmentation, follicular in character, involving the trunk and extremities. The circumoral region was spared. A netlike leukoplasic patch could be seen on the right buccal mucosa. There were no subjective sensations.

Her basal metabolic rate was —15 per cent. The sella turcica was normal on roentgen examination. A roentgenogram of the gastro-intestinal tract revealed colonic stasis with spasticity, and signs suggestive of a duodenal ulcer. The blood chlorides were 456 and blood sugar 92. The Wassermann reaction of the blood was negative. The blood picture was practically normal.

DISCUSSION

Dr. Parkhurst: The mottling was of almost a solid color, quite generalized, and the eruption was follicular. I found no keratoses on the hands or elsewhere, but think the case was of arsenic origin.

Dr. Ormsby: I wonder if Dr. White can tell us whether the pigmentation has been found below the epidermis. The color here presents the appearance presented when the pigment is situated in the corium.

DR. WHITE: I naturally watched Dr. Osborne's work and as I recollect it, the pigment never appeared below the epidermis. It was closely associated with the quintvalent arsenic deposits around the structures of ectodermal origin, especially the epidermis.

DR. BECKER: The girl presents patchy grayish pigmentation, which localizes the pigment in the superficial corium, as Dr. Ormsby mentioned. Two things produce this: first, ingestion of arsenic and, secondly, external irritation from tar products. I believe the history and histologic picture establish fairly well the diagnosis of arsenic pigmentation.

DR. WIEN: The sections for pigment staining are being run but are not yet completed. I will report on that and on the arsenic tests that are being made on the tissue at a later date.

NOTE: The microscopic sections were negative except for the presence of many large chromatophores containing tiny, round granules of deep brown pigment that filled the cell. A small number of the same kind of pigment cells were seen in the basal layer of the epidermis. The majority of these pigment cells were in the upper corium just beneath the epidermis. Pigment studies of the tissue by the method of Osborne, for the staining of arsenic in tissue, were negative for arsenic. The pigment in the sections being in chromatophores had the appearance and distribution of melanin.

Although this pigment is not arsenic, Gans has described anomalies in the distribution of melanin caused by arsenic without the presence of demonstrable arsenic in the tissues.

A CASE FOR DIAGNOSIS (LICHEN PLANUS?). Presented by DR. SENEAR and DR. WIEN.

A. C., a woman, aged 53, presented an eruption about the neck that had been present since August, 1928. It was preceded by a red, itching spot the size of a pea on the left breast which disappeared, and was followed by the neck eruption and a similar patch on the right side. There had been associated mild itching but no elevation of the lesions in the neck. Since the first of the year she had noticed that the eruption on the neck had been stationary and the past six weeks before presentation she had noticed a generalized eruption on the abdomen. She had recently suffered with nervous shock incidental to the illness of a son and daughter. There was no history of a previous skin eruption. Her general health was good.

The abdomen presented scattered shiny papules varying in size from that of a pinhead to that of a split pea, many of which exhibited striations on the surface and some of which were annular in configuration. The neck presented two areas of reticular atrophy interspersed through which could be seen shiny papules and follicular plugs. The area on the left side of the neck measured 2 by 12 cm. and that on the right 3 by 5 cm.

DISCUSSION

DR. PARKHURST: The lesions on the side of the neck with the reticulation and the follicular plugs were suggestive of lichen planus atrophicus. The lesions about the abdomen were flat, glossy, violaceous papules, quite characteristic of lichen planus.

DR. STILLIANS: The tendency of the lesions on the abdomen to become circinate bears out the diagnosis.

DR. ORMSBY: Occasionally lichen planus occurs with lesions that are followed by atrophy, but that is not lichen planus atrophicus. Lichen planus atrophicus is without color from beginning to end. One must recognize that there is atrophy occurring in ordinary lichen planus, but this is just as distinct as urticaria pigmentosa is from urticaria with pigmentation. I think this case is one of ordinary lichen planus with atrophy.

DR. SENEAR: I first saw this patient several weeks ago and she then had an eruption, as shown today, on the neck. We felt that it was lichen planus with atrophy. At that time there were not nearly so many lesions on the abdomen. They were acutely inflammatory, and I said then that there were two different lesions because the lesions on the abdomen were obviously excoriated. Today she has lesions on the abdomen which are flat-topped and I feel the condition is lichen planus and part of the neck.

NEUROFIBROMATOSIS. Presented by DR. STILLIANS.

An American woman, aged 22, a factory worker, when 7 years of age had first noticed "small warts" on the hand. They soon appeared elsewhere until at the age of 10 years they were generalized. Her general health was excellent.

Tumors were very profuse over the entire body, the largest being over 2 cm. in diameter. Others still larger had been excised. On the right cheek, left forearm and upper arm and on the anterior surface of the right thigh she had received one treatment with 25 mg. of radium at an inch distance for forty-eight hours on April 2 to 4, 1929. These areas showed distinct improvement. She was under treatment by Dr. Larkin. Her mentality was that of 13 years.

DISCUSSION

DR. ZEISLER: I recall one case that I saw a year or so ago in an institute for the feebleminded. An old woman had thousands of such tumors, some of the pendulous variety, and in addition she had an enormous sarcoma of the humerus.

DR. ORMSBY: It is interesting that this patient was presented by us before the Dermatological Society on April 17, 1924. At that time she was greatly disturbed mentally, and was put on some endocrine gland substance. In three months she reported that she was much generally improved, but there was no change in the condition of the skin.

DR. MITCHELL: I was told by a surgeon some time ago that he has seen a number of such cases and that no sarcoma of the bone has developed so far as he knows.

DR. FOERSTER: The bone tumors occasionally found in neurofibromatosis are often considered giant cell sarcomas, but pathologists prefer to call them giant cell tumors because of their relatively benign character as contrasted to sarcoma.

DR. MCEWEN: What type is her mentality now?

DR. STILLIANS: Her mental age is 13, although her chronological age is 22. I think, however, that this depends largely on who makes the test and the method used.

A CASE FOR DIAGNOSIS (TUBERCULOSIS OF SKIN?). Presented by DR. SENEAR and DR. CORNBLEET.

M. E., a white man, aged 52, had had a painful skin condition on the perianal region for six months before admission. It began as a chafe during a particularly warm day last August and the irritation was intensified by horse-back riding on the following day. From then on the lesion grew progressively worse until he was first seen, about six weeks ago. At that time he presented a shallow, smooth ulcer about 2 inches in diameter encircling the anus. The floor of the lesion was clean, exuded a glairy, foul-smelling discharge and was deeper on the left side. The left border was slightly thickened, and suggested the possibility of blastomycosis. All cultures and stained specimens of material from the border or base of the area had been normal. Wet dressings of potassium permanganate had been used, and there had been developing slowly a verrucous character to the lesion that was particularly evident over the left portion.

A biopsy specimen taken recently showed marked acanthosis; infiltration within the papillae of lymphocytes, macrophages, eosinophils and giant cells of the Langhans type. In some places there was an arrangement of many large macro-

phages that formed typical tubercles, in which were numerous Langhans giant cells. A few of the tubercles showed central necrosis. Acid-fast stains showed no organisms.

DISCUSSION

DR. MITCHELL: This was a very interesting case and I accept the diagnosis of tuberculosis without any hesitation.

DR. SENEAR: I remarked when showing the patient that in spite of the fact that this is the space where one expects tuberculosis and not blastomycosis particularly, the clinical picture when the patient was first seen was that of blastomycosis. Examination from that standpoint was completely negative, and when those things failed I proceeded in the search for tuberculosis. So far nothing has been found in the examination of the colon.

NOTE: Tubercle bacilli were later demonstrated in the biopsy specimen.

NEUROFIBROMA. Presented by DR. STILLIANS.

An American boy, aged 12, after an attack of acute encephalitis in about 1922, had an outbreak of nodules on the back. They gradually increased in number at the periphery of the patch with pigmentation and hypertrichosis.

The lesions were treated beginning in April, 1925, by the application of radium, from 200 to 800 mg. hours of radiation, with much improvement.

DISCUSSION

DR. OLIVER: This patient was presented to show the result of the radium therapy, and I think Dr. Larkin is to be much congratulated on the result obtained in the lesions about the neck and upper portion of the back. The boy's condition is markedly improved.

DR. STILLIANS: The diagnosis was made by a surgeon from a histologic examination. Many nodules have disappeared. The old pigmented area with hair on it looks as though there was a nevus there to start with.

DR. ORMSBY: We saw this patient in November, 1923. At that time he had a large pigmented hairy nevus covering the lumbar region and extending around to the groin laterally; also he had numerous tender nodules the size of hazelnuts in the lumbar region and flanks. In addition, there were large pigmented macules on the upper portion of the back. The diagnosis at that time was neurofibromatosis and nevus pilaris et pigmentosus. I note that the nodules described have disappeared during the interim.

DR. MITCHELL: The patient has improved greatly since I last saw him.

DR. OLIVER: I asked about the effect on the blood for I think it is of interest in treating such patients with large amounts of radium to have the condition of the blood carefully examined. I recently saw a baby who had been treated with the ordinary amounts of radium on the buccal mucous membrane. Just after the last treatment a severe anemia developed, which was blamed on the radium. The radium was not used in excessive amounts, however. I was interested to hear Dr. Larkin say that the blood count in this case was normal.

DR. STILLIANS: I had the same thing happen. A child was treated with a 5 mg. plaque for a hemangioma of the lip, and after several treatments developed an anemia that the pediatrician blamed on the radium.

DR. A. J. LARKIN: I did not think the radium was responsible for the anemia in the patient treated with the 5 mg plaque, but I questioned it in the other instance. If the treatments are given a month apart in a dosage kept under 2,000 mg. hours at a distance of 2 or 3 cm., there is relatively little danger. The white blood count is an indicator of the effect on the blood. If the count begins to go down it is better to wait from two to four months before continuing treatment. This applies to very young infants as well as to older children.

DETROIT DERMATOLOGICAL SOCIETY

H. J. PARKHURST, M.D., *Reporter*

Annual Meeting, May 1, 1929

GEORGE VAN RHEE, M.D., *Presiding*

LUPUS ERYTHEMATOSUS. Presented by DR. BEHN.

H. W., a man, aged 28, apparently in good general health, presented a sharply outlined erythematous patch on his left cheek the size of a coin, of three months' duration. The border of the patch was slightly infiltrated with an adherent scale, and the center was depressed. Immediately following a burn from a piece of hot steel, this lesion had appeared and gradually developed at the site of injury.

LUPUS ERYTHEMATOSUS. Presented by DR. SHAFFER.

S. Y., a man, aged 41, a merchant, born in Russia, had an erythematous plaque on the lower lip of eight years' duration. It began on the left side of the lip and spread slowly, involving the mucous membrane, vermilion border and adjacent skin, and reaching a diameter of 2.54 cm. Five years before presentation, roentgen treatments had been used, as indicated by an area of atrophy, with lesions of lupus erythematosus about its border and extending to the right side of the lip. There was only one other lesion: an erythematous, scaling patch, the size of a pea, on the chin. The Wassermann reaction was negative. There was one abscessed tooth and one devitalized tooth. The tonsils were not above suspicion. Roentgenologic studies revealed healed apical pulmonary tuberculosis.

LUPUS ERYTHEMATOSUS INVOLVING THE BUCCAL MUCOSA. Presented by DR. VALADE.

Miss B. S., a seamstress, aged 41, first noticed a lesion on the right ear in 1922, followed by involvement of the face. These lesions disappeared during the administration of gold sodium thiosulphate and did not reappear. Since the disappearance of the skin lesions, minute erosions had developed in the buccal mucosa, with thin whitish borders.

DISCUSSION

DR. JAMIESON: In the first case it is interesting to note that the eruption originated at the site of a burn from a piece of hot steel. In the second case the effects of gold therapy might be studied.

DR. SHAFFER: I have had a case like the first one, and the patient failed to respond to treatment with gold compounds. I wonder whether one is justified in administering gold in a case of a small lesion like this. It is a powerful drug, and the lesion might be removed readily with solid carbon dioxide. In the second case, which was mine, the patient has been rather a heavy smoker, and this, together with the atrophy following exposures to the roentgen ray brings up a possibility of future malignant degeneration. More roentgen treatment has been given recently, without response. I shall try gold therapy.

DR. LAVENDER: How was lupus vulgaris excluded in the second case?

DR. SHAFFER: Lupus vulgaris is not definitely excluded, although the lesion has been present for about five years, with practically no destruction of tissue, and the center seems typical of lupus erythematosus.

DERMOGRAPHISM, PITYRIASIS ROSEA AND PUNCTATE ANGIOMAS OF THE SCROTUM. Presented by DR. WOLLENBERG.

R. S. C., a man, aged 37, a ticket agent, since early childhood had noticed the appearance of wheals on the skin after scratching or stroking. About one month before presentation a small, slightly scaling, pink macule appeared near the right

groin. It slowly enlarged to the size of a large coin. Two weeks later, many similar lesions appeared on the trunk, arms and thighs, mostly on the abdomen and back. They were the size of a small coin and were slightly pruritic. For at least two years there had been many small punctate angiomas on the scrotum, some having a small dark center. None showed hyperkeratosis, but the patient stated that they had occasionally thickened, dried and peeled off.

DISCUSSION

DR. GUY: The scrotal lesions are of the type commonly confused with angiokeratomas. I should like to hear from Dr. Wile in regard to that point.

DR. WILE: The scrotal lesions, such as we have observed in this case, are in no way related to true angiokeratoma, which is a disease of the extremities, and which produces hemorrhagic lesions which become crusted and lead to atrophy. These scrotal lesions, which we commonly see, are merely nevoid structures. They are vascular lesions, analogous to the senile angiomas so commonly seen on the trunk. Histologically, and from the standpoint of their etiology, they in no way resemble true angiokeratoma.

DERMATITIS HERPETIFORMIS. Presented by DR. VAN RHEE.

Mrs. S. C., a widow, aged 65, born in Turkey, first noticed a burning and itching erythema on the trunk in December, 1928. About four weeks later, vesicles appeared on the arms and legs. There was intense pruritus. Six weeks before presentation there had been bullae on the face, neck, chest, back and extremities, including the palms and soles, and several lesions on the palate and buccal mucosa. There was definite grouping of the lesions, pigmentation and some scarring. The forearms and legs were crusted as a result of scratching. Nikolsky's sign was absent. Improvement resulted under treatment with potassium arsenite (Fowler's solution) and autogenous serum. One tooth was extracted. There was slight leukocytosis, and a trace of sugar was found in the urine.

NOTE: Subsequently a phytopharmacologic examination of the blood by Dr. Macht in Baltimore confirmed the diagnosis of dermatitis herpetiformis.

DISCUSSION

DR. WILE: Cases such as this, in which the diagnosis lies between pemphigus and dermatitis herpetiformis, are always worthy of serious discussion. Without having heard the history or knowing anything about the patient, I think that one would naturally be lead to the diagnosis of pemphigus. The blisters are somewhat larger than one ordinarily finds in dermatitis herpetiformis. The lack of erythema and the miserable appearance of the patient are consistent with the diagnosis of pemphigus. On the other hand, Dr. Van Rhee evidently observed this patient over a considerable period. There was a great deal of pruritus, which one does not ordinarily associate with pemphigus; but it sometimes does occur. I think this case belongs in a group often discussed in every dermatological society. In such cases it is impossible to say at any one time whether one is dealing with a herpetiform dermatitis of severe degree, or with pemphigus. If the patient ultimately dies, it is pemphigus. If he recovers, the diagnosis of dermatitis herpetiformis is accepted.

DR. GUY: I would suggest that in this questionable case we utilize the facilities at our command by sending a specimen of blood to Drs. Pels and Macht at Johns Hopkins for a phytopharmacologic test. They have shown rather definitely that in pemphigus the serum is changed and toxic, in contradistinction to other conditions, in which the serum is nontoxic.

DR. HOLLANDER: I wonder whether the question of dermatitis herpetiformis is applicable to this particular type of case when lesions are found in the oral mucosa. According to Oppenheim, the occurrence of mucous membrane lesions makes the possibility of dermatitis herpetiformis much more remote. This patient has definite mucous membrane lesions. She is a Jewess, and at the age when

pemphigus commonly appears. Under these conditions, I wonder whether a diagnosis of dermatitis herpetiformis should be considered.

DR. FISHER: This eruption is generalized, with no grouping, and with oral lesions. I favor the diagnosis of pemphigus and would advise the Davis treatment. I used that treatment in a case which I presented before the Cleveland Dermatological Society a few years ago. For the past year and a half the patient has been free from lesions and able to work.

DR. VAN RHEE: My experience with the Davis treatment has been limited. I have tried it in two cases, and both ended fatally. I am willing to try it in this case.

DR. TAUBER: I have used it in about nine or ten cases.

GENERALIZED DISCOID LUPUS ERYTHEMATOSUS. Presented by DR. SCHILLER and DR. SCHLEGELMILCH.

Mrs. N. A., a negress, aged 38, had a genital lesion ten years previously, at which time she received antisyphilitic treatment for three or four months. Since that time, repeated Wassermann tests were negative. She had never been pregnant. About a year after the antisyphilitic treatment, severe pruritus developed in the genital region; it persisted until the time of presentation. Two years later the neck and scalp began to itch severely, and an eruption appeared on the nape of the neck, involving the scalp, with loss of hair, and extending to the chest, arms, back, abdomen and thighs, in the order mentioned. The scalp was almost totally bald, with scaling macules and atrophic areas. Over the chest there were single and confluent macules with depressed, hyperpigmented centers. The margins were raised and irregular. The lesions on the back presented marked pitting. The eruption involved the extensor surfaces of the forearms, shoulders, back, buttocks and thighs. The labia were greatly thickened and discolored.

DISCUSSION

DR. WILE: In my experience, at least, this is an unusual case, with the extraordinary dissemination and uniform character of the eruption. I do not think one would be justified in calling it lupus erythematosis disseminatus. I think it is all discoid lupus erythematosus, precisely of the kind one ordinarily finds on the sides of the face and the bridge of the nose. I do not think I have ever seen it as widespread as this before. Cases of the disseminated type do not look like this. Its chronicity is unique. I should like to hear from some of the men living in urban communities regarding their experience with cases of this kind.

DR. OSBORNE: I should like to mention two cases. One was presented before the Chicago Dermatological Society last year by Dr. Ormsby. The patient was a blond woman with an eruption of the chronic type, a little more widespread than in the present case, even the soles of the feet being involved. There is another case which the late Dr. Wende observed for five years, with the most extensive eruption of this type that I have ever seen. Within the past two or three years he has had, in addition, two or three acute exacerbations of the acute disseminated type. Several years ago, I saw a patient of Dr. Schamberg's: a woman with more than 300 such lesions scattered over her body. So far as I know, none of the patients in these cases has shown improvement under treatment with gold therapy, even with a large number of injections.

ATROPHIC SCARS FOLLOWING A SECONDARY SYPHILID. Presented by DR. SCHILLER and DR. SCHLEGELMILCH.

In A. L., a negress, aged 26, syphilis developed two years previously, with a generalized eruption. This rash disappeared, leaving small atrophic scars, varying in size from that of a pinhead to that of a pea, which appeared as flaccid projections. There was also a generalized eruption of small pinkish and purplish papules, ovoid and rather scaly. There were signs of pulmonary tuberculosis

and tuberculous cervical lymphadenitis. The Wassermann reaction was negative. Under ultraviolet irradiation of the tuberculous lymph nodes, a large number of discrete pinkish papules developed on the chest and back.

DISCUSSION

DR. DRIVER: In clinics in which a large number of negroes are seen, this type of scar following secondary syphilis is not unusual. I have seen it frequently. Three years ago, before the Cleveland Dermatological Society, I presented an extensive case of this type.

SPOROTRICHOSIS (?). Presented by DR. JAMIESON.

J. F. K., a man, aged 64, a clerk, whose history seemed irrelevant to the condition presented, first noticed a small suppurative lesion, following a slight injury, on the terminal phalanx of the left index finger six months previously. It slowly developed into a granulomatous lesion, slightly purulent. A week or two later there appeared two small erythematous subcutaneous nodules on the forearm in the line of drainage, and also lymphadenitis in the cubital and axillary regions. The Wassermann reaction was negative. A guinea-pig was inoculated with material from a subcutaneous nodule, but only staphylococci were recovered. Under various treatments the initial lesion became flattened. The nodules on the forearm had suppurated, and had not healed at the time of presentation. Lipoiodine was administered for two weeks without much effect.

DISCUSSION

DR. JAMIESON: The condition has partly subsided, and there are now few active lesions. Dr. Valade saw the patient before I did, and at that time the original lesion was like a pyogenic granuloma. I first saw him a month or two later, after the nodules and lymphadenitis had appeared. He has not been in contact with rabbits. The chronicity of the condition and the involvement of the areas in the line of drainage, in spite of the fact that no organisms were found, point to a possible diagnosis of sporotrichosis.

DR. OSBORNE: I should like to suggest the possibility of inoculation tuberculosis. The original lesion on the finger started in a manner rather typical of tuberculosis. It would more probably be due to the bovine than to the human type of tuberculosis.

DR. NETHERTON: I would suggest that a test for tularemia be made. This may be a case of the ambulatory type.

ACTINOMYCOSIS (?). Presented by DR. VALADE.

In J. A., a Mexican laborer, aged 40, abscesses developed with sinus formation in the cervical region five years previously while he was doing railroad work in Kansas. These sinuses healed, and new glandular abscesses appeared gradually. Two or three years before presentation, in Detroit, a diagnosis of tuberculosis was made. There were many scars of healed sinuses about the neck and upper part of the trunk, and discharging sinuses in both axillae, the right inguinal region and the scrotum. Smears and cultures were negative, and a guinea-pig was inoculated two weeks previously, with no apparent result. A histologic diagnosis of tuberculosis was made, and roentgenograms showed infiltration of the right lung, but no active tuberculous process. The Wassermann reaction and urinalysis were negative. A blood count revealed an increase in polymorphonuclear leukocytes.

ACTINOMYCOSIS (?). Presented by DR. VALADE.

V. A., a Mexican woman, aged 30, a sister of the preceding patient, had a small axillary glandular abscess seven or eight months previously, which was incised. A discharging sinus persisted. There had not been sufficient time for further investigation in this case.

DISCUSSION

DR. WILE: I have studied the sections and consider the picture that of actinomycosis, in agreement with the clinical observations.

A CASE FOR DIAGNOSIS. Presented by DR. STEVENS and DR. HASLEY.

A. F. T., a machinist, aged 24, of anemic appearance, said that he had had a dry cough and had lost a few pounds in weight during the preceding two years. There had been no fever nor night sweats. In January, 1927, after a year of "chronic bronchitis," the patient first noticed nodules at the vertex of the scalp; they enlarged gradually. The right shoulder then became painful, and in April, 1927, a large mass appeared on its posterior aspect. In July, 1927, all the lesions were incised, and large amounts of thick pus were removed. The scalp then healed, but small nodules the size of a pea persisted at the occiput, with a slight purulent discharge. Several discharging sinuses persisted on the shoulder. The patient then complained of "sciatica," and in March, 1928, a large tumor appeared on the left buttock. It was incised and cultures were made on plain and Sabouraud's mediums, with no important observations. In June, 1928, a line of small nodules appeared in the left supra-orbital region. In September, 1928, roentgenograms showed a shadow, larger than a goose egg, rounded and sharply circumscribed, apparently in the lateral posterior pleura, projecting into the base of the right lung. It was diagnosed as an encapsulated abscess. In January, 1929, a large doughy mass, 6 inches (15 cm.) in diameter, developed on the right buttock, and somewhat later a tumor, about 2 inches (5 cm.) in diameter, appeared on the left shoulder. Cultures made at several times on plain and Sabouraud's mediums, with material aspirated from tumors or taken from open sinuses, revealed nothing. Sections were made from a lesion on the left buttock, and epithelioid and plasma cells, lymphocytes and giant cells were found; no fungi were seen. Beginning in September, 1927, as much as 250 grains (16.25 Gm.) of potassium iodide was given daily, but there was no improvement until roentgen treatment was started. Several sinuses then healed, and the development of tumors in the right buttock and shoulder was apparently arrested.

DISCUSSION

DR. STEVENS: I have considered this a possible case of actinomycosis, involving the lung. I feel that no roentgenologist would call the pulmonary lesion tuberculous.

DR. WILE: Probably the diagnosis will remain obscure until the nature of the pulmonary lesion is determined.

(Subsequently, tubercle bacilli were found in inoculated guinea-pigs.)

ACTINOMYCOSIS (?). Presented by DR. BEHN.

I. F., a man, aged 26, first noticed a swelling of the left part of the lower jaw four months previously. Two carious teeth were extracted, and the pain and swelling increased. He then entered Massachusetts General Hospital, where a diagnosis of actinomycosis was made from cultures and smears. Dr. Behn first saw the patient one month before the time of his presentation, with an indurated mass involving the jaw and neck. There were five sinuses in this mass, discharging a thin, flocculent pus. Rapid improvement followed the administration of large doses of potassium iodide and four roentgen treatments. The causative organism was not demonstrated; the Wassermann reaction was negative.

TUBERCULOSIS CUTIS; LATENT SYPHILIS. Presented by DR. VALADE.

V. B., a negress, aged 33, a widow, had received antisyphilitic treatment seven or eight years previously. After the second or third injection of arsphenamine, an eruption of large annular papules and nodules appeared on her face and developed over a period of two years. At the end of this period, six injections of arsphenamine and twelve of mercury produced no change in the lesions. The eruption

had extended, and at the time of presentation the neck and arms were also involved. There had been no ulceration, but a number of thin scars were present. Some of the lesions on the face had raised borders and follicular plugs, suggesting lupus erythematosus. Several finger joints were swollen. The Wassermann reaction had been strongly positive. A diagnosis of tuberculosis had been made after a biopsy examination at the University of Michigan.

DISCUSSION

DR. VALADE: Following twelve injections of a gold compound I saw no improvement in the lesions.

DR. BELOTE: This patient was first seen in the clinic at Ann Arbor eight or nine years ago, and at that time she had no cutaneous lesions. She was treated for syphilis, and during the course of treatment lesions appeared on the skin. Some of the lesions are typically tuberculous, but others suggest lupus erythematosus.

TUBERCULOSIS CUTIS. Presented by DR. SCHILLER and DR. SCHLEGELMILCH.

Mrs. M. S., a negress, aged 26, presented a generalized eruption of small papules, some showing central involution and scaling. The eruption first appeared on the face two years previously, and had been generalized only three or four months. Depigmented scars marked the sites of previous lesions. A histologic diagnosis of tuberculosis was made. Although the Wassermann reaction was strongly positive, there was no response to antisyphilitic treatment.

LUPUS VULGARIS. Presented by DR. BEHN.

G. S., an Austrian, aged 45, a laborer, first noticed a small papule or pustule under his chin four years previously. It persisted and enlarged. It was violaceous or reddish brown, and markedly infiltrated, especially about the border. There was considerable crusting. Various local applications, including solid carbon dioxide, had brought no relief. The Wassermann reaction was negative. Gold sodium thiosulphate was being administered.

SCHAMBERG'S PROGRESSIVE PIGMENTARY DERMATOSIS. Presented by DR. TROXELL.

A. E. B., a man, aged 34, a bookkeeper, had had varicose veins in his legs for seven years. In 1920, pigmentation over the shins was noted. Since that time it had slowly progressed and extended. There were a number of reddish-brown patches varying in size from that of a pea to that of a medium sized coin, sharply outlined, surrounded by many brownish macules, the size of a pinhead interspersed with reddish punctae. There had been no ulceration and no subjective symptoms.

SCHAMBERG'S PROGRESSIVE PIGMENTARY DERMATOSIS (?). Presented by DR. VALADE.

J. P., a mechanic, aged 29, presented an eruption of large and small brownish and purplish macules, with telangiectases and cayenne pepper-like spots on the arms, trunk, thighs and legs. It began two years previously as a scaling dermatitis. There was an 8 per cent eosinophilia.

DISCUSSION

DR. PARKHURST: In Dr. Troxell's case the slate-colored, somewhat infiltrated plaques about the ankles were rather suggestive of Kaposi's sarcoma.

DR. HOLLANDER: Dr. Valade's patient presented pigmented lesions with slight atrophy on the legs and arms, and one on the abdomen. He works with molten metal, and states that it splashes on the affected parts. It may be an industrial dermatosis.

DR. LAVENDER: I agree with Dr. Hollander, although the case presents some points suggestive of acrodermatitis chronica atrophicans.

LICHEN PLANUS ANNULARIS. Presented by DR. TROXELL.

F. R., a man, aged 44, a purchasing agent, first noticed papules the size of a pinhead on the wrist flexures three months previously. These lesions increased rapidly in size and number, involving the extremities and trunk. The larger lesions were well defined, circular or oval, with a threadlike elevated margin and a smooth center of atrophic appearance. There were no subjective symptoms.

LICHEN PLANUS LINEARIS. Presented by DR. SHAFFER.

A. B., a man, aged 36, stated that three months previously he had first noticed a markedly pruritic bandlike eruption along the left lower rib margin. Soon afterward a second band developed about 3 inches below the first, being followed shortly by a third on the left flank. These bands had a curved linear distribution roughly approximating that of the nerves, with a superficial resemblance to zoster. They were made up of closely set, flat-topped, angular purplish papules, with a rather unusual amount of scaling. There was also a chronic erythematous eruption on both cheeks, a left corneal ulcer and a recurrent pruritus ani.

ADENOCARCINOMA OF SWEAT GLAND ORIGIN. Presented by DR. STEVENS and DR. HASLEY.

Mrs. S., aged 34, born in Scotland, stated that she had had a lump in the right cheek since 1920. Following an operation in Scotland, in 1921, the condition was diagnosed as tuberculous. The patient first saw Dr. Stevens in August, 1923; she then had a hard, lime-sized lump in the cheek, suggesting a parotid tumor. Since that time roentgen and radium therapy had been used at intervals. In February, 1927, a discharging sinus was found in the roof of the mouth, leading to the tumor. Following radium applications in March, 1928, the tumor had almost completely disappeared, only to recur by gradual enlargement. It was hard, nodular, scarcely movable and apparently not connected with the parotid gland. A biopsy examination revealed an adenocarcinoma of sweat gland origin and a basal cell carcinoma.

A CASE FOR DIAGNOSIS. Presented by DR. JAMIESON.

M. McC., a Scotch girl, aged 16, a student, for eight summers had had lesions on the parts of the face and extremities exposed to the sunlight. The upper parts of the chest and back were also involved. The outbreak presented, of two months' duration, consisted of pruritic, widely scattered, small, discrete, excoriated erythematous papules. At the tip of the nose there was an area suggesting lupus erythematosus. The condition had been less pronounced when the patient had lived in northern Canada. No hematoporphorin was found in the urine.

DISCUSSION

DR. PARKHURST: The lesions seem to be limited to the areas which are exposed to the light, and in view of the comparative absence of scarring from previous lesions and the mildness of the eruption, I should consider this a case of Hutchinson's summer prurigo.

DR. GUY: I agree with that diagnosis.

LICHENOID PARAPSORIASIS. Presented by DR. VAN RHEE.

Mrs. B. S., born in Austria, first noticed an eruption on the trunk and arms three years previously. It extended to the lower extremities, without any subjective symptoms. The eruption had been present constantly since the onset. There was a widespread papular eruption, some of the lesions being suggestive of lichen planus while others were scaling and resembled psoriatic lesions.

ACANTHOSIS NIGRICANS. Presented by DR. JAMIESON.

Mrs. D. C., aged 33, born in Hungary, was the mother of four children. She had been doing factory work for a year, during which time there had been a gradually increasing feeling of lassitude. For five months ill defined blackish areas had been developing on the face, neck, arms and axillae, with some velvety thickening of the skin. The blood pressure was 110 systolic and 70 diastolic.

DISCUSSION

DR. WILE: This seems to be a typical early case of acanthosis nigricans. In the axillae and about the neck the hyperpigmented skin has a velvety appearance. Whether a neoplasm will develop, or whether this case is of the juvenile type, will be a matter to determine.

DR. WOLLENBERG: Although I agree with Dr. Wile's remarks, I feel that the possibility of Addison's disease must not be overlooked. The patient, however, does not seem to have the usual degree of asthenia.

DR. HOLLANDER: There is no hyperpigmentation about the nipples such as would usually be found in Addison's disease, and I think that diagnosis can be excluded. These lesions developed while the woman was working with lacquer. Pruritic erythematous areas were said to have appeared on the nose and neck, becoming brownish after repeated excoriation. This may be an industrial dermatosis.

DIFFUSE SYMMETRICAL SCLERODERMA. Presented by DR. SCHILLER and DR. SCHLEGELMILCH.

Mrs. F. H., aged 55, had noticed cutaneous hyperesthesia of the neck a year previously. Two or three months later the skin of the neck became stiff, so that she had difficulty in moving her head. There were pains in the joints and the fingers became stiffer than normal. A hyperpigmentation appeared on the face, neck and extremities, in large and small patches. The skin of the neck and upper part of the midchest was hard, boardlike and waxy, with irregular pigmented and atrophic areas. There was an erythematous margin about this area. The skin of the feet, legs, thighs, hips, arms and face presented a hide-bound appearance, and there were small atrophic areas scattered over the entire body. There was no loss of sensation.

SCLERODERMA. Presented by DR. SCHILLER and DR. SCHLEGELMILCH.

Mrs. R. S., aged 44, had been in comparatively good health, but had had a double oophorectomy four years previously. Five years before the operation, a band of hyperpigmentation appeared, encircling the neck and extending upward to the ears and downward to the upper part of the sternum. There were burning sensations and the skin was thickened. The neck movements were hampered.

MORPHEA. Presented by DR. HYDE.

Mrs. M. H., aged 31, first noticed a red spot the size of a pea on the right upper arm about four years previously. It had reached the size of a large coin and persisted. About one year before the time of presentation a cordlike band began to develop, extending upward from the lesion toward the shoulder. The original patch was ivory-colored, with a violaceous border, and the band extending to the shoulder was violaceous.

MORPHEA. Presented by DR. STEVENS, DR. JARRE and DR. HASLEY.

Mrs. J. S., aged 28, first noticed a patch on the right thigh in June, 1928. It was gradually extending. She was in the second month of pregnancy.

DISCUSSION

DR. WILE: Dr. Stevens' case brings up the question as to where morphea ends and scleroderma begins. The lesions of morphea are usually not linear, as these are. They are more likely to be round or oval, and seldom show the degree of sclerosis found in this case. I consider this a case of scleroderma.

DR. GUY: In Dr. Schiller's patient with the involvement of the neck, I felt that the condition was a chronic inflammatory process, not scleroderma.

DR. HOLLANDER: The case Dr. Guy referred to impressed me as one of toxic erythema rather than scleroderma.

DR. BUSMAN: The history of five years' duration, the complaint of stiffness of the neck and the history of oophorectomy preceding the condition, all lend support to a diagnosis of scleroderma.

PERIFOLLICULITIS CAPITIS ABSCEDENS ET SUFFODIENS. Presented by DR. SHAFFER.

S. R., a man, aged 26, a laborer, stated that the condition began two years previously with lesions on the vertex of the scalp. It slowly extended, involving the entire scalp, especially the vertex and occiput. The active lesions were nodules, varying in size from that of a pea to that of a hickory nut, some firm, others fluctuating, and varying from pinkish yellow to a livid hue. The healed lesions had left irregular depressed scars, some being verrucous and irregularly elevated. There were also many papulopustular acneiform lesions on the face and a few on the back, together with small pitted scars. According to the history, the usual antiseptic applications had been of little help. Late in 1928 the patient was under observation for six weeks at Ann Arbor.

DISCUSSION

DR. PARKHURST: This seems to be a case of perifolliculitis capitis abscedens et suffodiens, although the eruption on the face and back suggests the acne conglobata of Lang. I feel that the condition is probably of staphylococcic origin.

DR. GUY: I agree with the diagnosis of the case as presented. Perhaps roentgen treatment would be effective.

DR. BARNEY: I have had under observation for the last three or four months a typical example of this disease. I have studied it rather thoroughly and taken several biopsies, each showing a granuloma-like reaction. Repeated cultures have revealed only the staphylococcus. There has been some improvement following treatment with roentgen rays and intramuscular injections of foreign protein.

DR. PHILLIPS: I had a typical case of this condition, in which I administered one-fourth skin unit of roentgen rays every other week, alternating with erythema doses of ultraviolet rays. After twelve treatments the cure has been complete.

ANNULAR AND FOLLICULAR SECONDARY SYPHILID. Presented by DR. WILE.

E. D., a man, aged 21, a truck driver, had a primary sore two years previously, with involution under treatment with nine injections of neoarsphenamine. He received no other treatment. At that time there were no secondary manifestations. Six months before the time of presentation he became hoarse, the hoarseness persisting. Two months later he noticed the appearance of an eruption, with no subjective symptoms. There was a generalized eruption, the scalp and scrotum being involved. The hair of the entire scalp was thinned and lacked luster. Over the forearms, thighs and scrotum, especially, there was a follicular and annular papular and papulosquamous eruption, of a dull brownish red. The annular lesions were the size of a small coin, and the follicular papules were the size of a pinhead. The eruption was not pruritic. There was a generalized lymphadenitis, including both epitrochlear lymph nodes. On the dorsum of the shaft of the penis there was an atrophic scar the size of a pea.

DISCUSSION

DR. TAUBER: This type of eruption is frequent in the colored race, the lesions occurring mostly on the face. The appearance of this patient's nails suggests that he may have some colored blood.

ATROPHY OF THE FATTY LAYER OF THE SKIN (GILCHRIST AND KETRON). Presented by DR. SCHILLER and DR. SCHLEGELMILCH.

B. S., a widow, stated that about twelve years previously she had noticed a thickening of the skin of the right arm, followed by a shrinkage of the tissues. These areas appeared with more or less regularity for a considerable period. In April, 1916, a tentative diagnosis of morphea was made at the Mayo Clinic. There were numerous large and small morphea-like patches on the arms, shoulders and cheeks, showing a considerable loss of subcutaneous fat. A few months before the time of presentation an eruption of lupus erythematosus developed on the face, ears, scalp and fingers; it faded subsequently.

LICHEN ACUMINATUS (?). Presented by DR. BIDDLE.

B. B. G., a Jew, aged 38, an attorney, had an eruption of dry, scaling, shiny lichenoid papules on the middle third of the left leg for eight years, with occasional severe pruritus. More recently the other leg became affected in a milder degree. Pigmentation became gradually more pronounced.

DISCUSSION

DR. GUY: In resistant cases such as this, the following procedure may be helpful: thoroughly cleanse the area with ether, then paint it with a strong solution of silver nitrate and immediately oxidize it with an erythema dose of ultraviolet rays.

MADURA FOOT (?). Presented by DR. JAMIESON.

S. M., an Italian, aged 38, a grocery clerk, was presented before the society two years previously with a swelling of the foot and leg showing numerous abscesses and sinuses. The picture then was that of a Madura foot. The pathologic diagnosis was tuberculosis, but no organisms were found with the microscope nor on inoculation or cultures. During the two years since the previous presentation, large doses of potassium iodide were given, and some ultraviolet ray treatments. Improvement was steady, with temporary relapses. The abscesses disappeared, and the sinuses practically stopped discharging.

DISCUSSION

DR. TAUBER: I am inclined to agree with a diagnosis of Madura foot in this case.

POIKILODERMA ATROPHICANS VASCULARE (?). Presented by DR. WILE.

Mrs. D. W., aged 68, first noticed a burning and itching edema about the eyes four years previously. Shortly afterward, she noticed similar sensations about the face and neck, gradually spreading over practically the entire body. In addition, she noticed considerable redness and scaling, particularly over the extremities. Two years before the time of presentation she noticed several cutaneous nodules over the buttocks. During the course of the disease she had lost 40 or 50 pounds (18.1 or 22.7 Kg.), but had recently regained some of it. Her general physical condition was fairly good. There was a definite history of frostbite during the patient's early youth. Otherwise the history seemed irrelevant.

There was a generalized eruption with a predilection for the extensor surfaces of the extremities and the trunk, face and neck. It consisted of an underlying purplish erythema, scattered throughout which were occasional areas of almost normal skin. Considerable fine, flaky, branny exfoliation was present. Also there

was a marked dilatation of the superficial capillaries, and scattered throughout the involved regions there was a diffuse atrophy of the skin. Circumscribed areas of normal skin were noted about the umbilicus and nipples. Over the right buttocks were about twelve deep-seated nodules, discrete, firm, and not tender. There were no evidences of internal pathologic changes, and the blood and urine were within normal limits. Special studies of the blood chemistry revealed nothing of interest. The sella turcica was small.

DISCUSSION

DR. WILE: This elderly woman has an extraordinarily uniform eruption which closely resembles "x-ray skin." The condition has not changed during the two years I have observed her. There have been occasional periods of pruritus. She has been seen by members of the Chicago Dermatological Society, and a number of able observers have suggested a diagnosis of poikiloderma.

CASE FOR DIAGNOSIS. Presented by DR. VALADE.

R. S., a Polish housewife, aged 51, for at least seven months had had a gradually extending violaceous group of nodules the size of a pea on the right ala nasi, extending into the anterior nares. There was considerable infiltration. The Wassermann reaction was negative.

DISCUSSION

DR. GUY: The case impressed me as a granuloma: either tuberculosis or syphilis.

CASE FOR DIAGNOSIS. Presented by DR. WOLLENBERG.

Mrs. J. C., a Jewess, aged 43, had continuous redness, itching and burning of the cheeks for five years. There was slight infiltration, a few papules, and slight crusting and scaling. Neither rouge nor face powder had been used for several years. There was marked constipation and mild dysmenorrhea. There were two old tooth roots in the upper jaw, and infected tonsils.

DISCUSSION

DR. GUY: I consider this a case of rosacea. There is a history of digestive disturbances, and I would suggest that gastric analyses be made.

A CASE FOR DIAGNOSIS: XANTHOMA (?). Presented by DR. JAMIESON.

J. S., a boy, aged 6 months, presented a yellowish-red nodule the size of a small coin on the inner side of the left thigh, just above the knee. The nodule was intracutaneous and flat topped, with an underlying and surrounding infiltration. It had been present at birth, and when first seen by Dr. Jamieson, three months before the time of presentation, it had a slightly elevated reddened border and a somewhat depressed center of lighter color. During the three months of observation it had been undergoing gradual involution.

DISCUSSION

DR. WILE: This appears to be a case of xanthoma. At the Ann Arbor meeting of the Chicago Dermatological Society in February, 1929, I presented a case in a child well under 1 year old, the lesions having appeared a month after birth. It is also possible to have a solitary lesion. We had a student at the university a few years ago with a single large xanthomatous lesion on the buttock.

GUMMA OF THE TONGUE. Presented by DR. VAN RHEE.

Mrs. C. C., had had a medium coin-sized nodular and ulcerative lesion on the tongue for six weeks. There was slight infiltration and the cervical lymph nodes were palpable. The Wassermann reaction was strongly positive. A biopsy specimen was presented.

BROMIDROSIS; HYPERHIDROSIS; LIVIDITY OF THE SOLES. Presented by DR. JAMIESON.

A young man, a student, presented symmetrical sodden, whitish areas on both heels and a part of each sole, with pinkish borders. The perspiration was excessive and malodorous.

DISCUSSION

DR. WILE: This condition may be associated with a mild type of frostbite.

DR. WOLLENBERG: I feel that the condition is the result of hyperhidrosis, and would suggest applications of a 30 per cent solution of aluminum acetate.

DR. BEHN: I would suggest painting the foot every night with a strong solution of potassium permanganate and removing the stain every morning with oxalic acid solution.

ROUND CELL SARCOMATOSIS OF THE NEW-BORN. Presented by DR. STEVENS, DR. JARRE and DR. HASLEY.

A negro baby died at the age of 1 month. It was born at full term, was well developed and weighed over 9 pounds (4.1 Kg.). The parents were healthy, and there were no evidences of syphilis. Six days after birth an examination revealed a generalized eruption of discrete and confluent papules and nodules. Some were waxy, but the smaller lesions were pinkish to rose colored, blanching under pressure. The skin showed little pigment. Two weeks later the skin became infiltrated, and death rapidly ensued. The diagnosis was based on postmortem microscopic studies made at Ann Arbor. Photographs and slides were presented.

CASE FOR DIAGNOSIS: PARAPSORIASIS (?). Presented by DR. TROXELL.

Mrs. S. M. T., aged 51, first noticed a dermatitis on the face and neck in October, 1924. Rapidly becoming generalized, this condition was followed by profuse exfoliation for several months. In March, 1925, small acuminate papules appeared about the hair follicles of the arms and legs; this eruption soon became generalized, coalescing on the arms and legs to form erythematosquamous areas. This condition lasted a year and was followed by marked exfoliation and complete disappearance of the horny plugs. During all this time there had been intense pruritus. The exfoliation gradually diminished, leaving a marked erythema and some epidermal thickening. Following this, the patient was not seen for two years. At the time of presentation there was a retiform eruption over the trunk and extremities, with slight scaling in some areas. The subjective symptoms had largely abated, and the general health was much improved.

The patient was presented before the society in April, 1925, and in May, 1926.

ST. LOUIS DERMATOLOGICAL SOCIETY

NORMAN TOBIAS, M.D., *Secretary*

Regular Meeting, May 8, 1929

ROBERT H. DAVIS, M.D., *President*

A CASE FOR DIAGNOSIS. Presented by DR. JOSEPH GRINDON.

A white man, aged 26, a student of large frame, muscular and in excellent health presented a small nodule below the right scapula. Most of the time the lesion presented the appearance of an ordinary pigmented mole, unaccompanied by subjective symptoms, but about once a month it passed through a cycle of curious changes occupying about seven or eight days. I observed the lesion in various stages, and through one complete cycle.

On January 29, I first saw the patient. The lesion was then in the quiescent stage, so that I confess I somewhat doubted the patient's account of its strange metamorphoses. On March 4 and 5, I saw it while active and its appearance on each of those days was exactly the same as on corresponding days of the complete cycle to be described later. On April 4, the patient was seen by Dr. Kring, when the appearance of the lesion was similar to that on March 4.

I again saw the patient on April 26. The cycle had commenced anew on the preceding day. There was a central molelike papule, slightly elevated, circular in outline and 4 mm. in diameter. Within it were three dots, almost black. Surrounding it was a disk, apparently purpuric, 1 cm. in diameter. These measurements were identical with those taken on March 4. The lesion was not painful, but tender to slight pressure. It never bled. On the 27, the lesion was less tender. The enclosed black dots were a little larger. On the 29, the lesion was not tender, but itched slightly. The central lesion was almost flat, the enclosed dots had coalesced and seemed to have risen to the surface so as to form an attached smooth, minute, black scale. The outer ring or halo was of lighter shade, fading at the periphery. On May 1, the central black dots were easily picked off as a thin scale, leaving only a minute central nonpigmented, somewhat reddened macule. The hemorrhagic halo, as well as all subjective phenomena, had disappeared.

The patient said that this cycle has been repeated seven times.

DISCUSSION

DR. BROCKELMANN: I think I have read something of the different appearances which nevi undergo.

DR. GRINDON: When quiescent it looks like a little flat, pigmented mole, an insignificant lesion the size of a pinhead.

DR. TOBIAS: The only way I can explain the cyclic character of the lesion is that it is due to trauma from resulting inflammation.

DR. DAVIS: It looks like a superficial malignant condition.

DR. GRINDON: It stays away about three weeks and then comes back. The only thing that becomes raised is the little central mass, which at each of two different cycles grew to 4 mm. in diameter. Around it is a little border of purpuric character, which measures just 1 cm. I do not know that I have seen anything like it at any time. There seems to be a little intradermal hemorrhage occurring at intervals of about four weeks.

ROENTGEN DERMATITIS. Presented by DR. ERICH BROCKELMANN.

A white woman, aged 26, single, always healthy, had roentgen treatments two years previous to presentation (Tricho system) for epilation of the hairs on both arms. She received six treatments (two weeks apart) when she noticed swelling and pain in the right forearm, which disappeared shortly afterward.

The patient presented multiple telangiectases on both forearms and lower parts of the upper arms varying in size from that of a pinpoint to that of a split pea and larger. The lesions were not elevated above the surrounding skin; they showed dendritic ramifications and were so numerous that they showed a retiform mottling of the skin. On pressure they disappeared completely to reappear after the pressure was released.

DISCUSSION

DR. TOBIAS: It is a wonder that we do not see more cases of this type, considering the number of quack concerns permitted to use the x-rays on patients with hypertrichosis.

DR. BROCKELMANN asked the patient why she had denied previous questions about the use of x-ray, and she stated that she was ashamed to admit it.

DR. GRINDON: Many years ago when the gas tube was first used, it was thought to be a fine thing for the removal of superfluous hair. I had a woman

patient, aged 27, from whom I removed facial hair by this means. Since then she has been coming back to see me once in a while with atrophy and numerous telangiectases of the face and neck.

LATE SYPHILIS OF THE TONGUE. Presented by DR. M. C. SNIDER.

A white man, aged 21, gave a history of a penile lesion three years previous to presentation for which he was given six injections into the arms; he did not recall whether they were intravenous or intramuscular. He denied the presence of an eruption on the body. He was admitted to the clinic for a sore tongue of seven weeks' duration.

The patient presented an indurated ulcer on the dorsal surface near the tip of the tongue. The Wassermann reaction was not reported.

DISCUSSION

DR. ENGMAN, JR.: The site in this case is an unusual place for carcinoma. Syphilis should be considered strongly.

DR. LANE: The history of a penile lesion, with intravenous treatment three years ago suggests a history of syphilis. The present lesion looks syphilitic and the jagged teeth on which the tongue rubs may have caused trauma, thus accounting for the location of the ulcer.

DR. GREINER: It looks like the result of ordinary trauma to me.

DR. TOBIAS: Although the dark-field examination gave negative results, I think a therapeutic course of antisyphilitic treatment would settle the diagnosis. Is the patient epileptic? I ask the question because there is a short cut on the tongue which might occur in an epileptic seizure.

DR. GRINDON: It might also occur from an accident.

DR. DAVIS: I thought it was a lesion due to trauma.

INFANTILE PSORIASIS. Presented by DR. M. C. SNIDER.

A white boy, aged 4, was admitted to the clinic with a history of an eruption on the extremities and body of six months' duration. The father had an eruption on the elbows which appeared every fall. Examination presented erythematous silvery scaled lesions, distributed on the extensor surfaces of the forearms, legs, buttocks and back. There was a tendency toward annular arrangement.

DISCUSSION

DR. SNIDER: It is a typical case of psoriasis, unusual in a child 4 years old.

DR. GRINDON: I have seen the condition in children that age and younger. George H. Fox, in his work on skin diseases of children, has a frontispiece showing a young child with psoriasis.

PITYRIASIS RUBRA PILARIS. Presented by DR. W. W. COULTER.

A white woman, aged 26, married, with a negative past, family and personal history, entered the clinic complaining of "eczema all over" of seven months' duration. There were no associated symptoms. The eruption was symmetrical and involved the neck, chest, abdomen, buttocks, thighs and arms to the elbow joints and consisted of red tinted, superficial, slightly scaly sheets. At the edges of the sheets there were scattered follicular maculopapules. There was considerable scaling of the scalp, hyperkeratosis of the palms and soles and atrophy of the finger-nails. There was a slight roughening of the extensor surfaces of the forearms but no apparent lesions.

Results of examination of the scales and cultures were negative for mycelia and spores. The blood and urine were normal, and physical examination gave negative results. The Kahn reaction was negative. Observations at biopsy were not reported.

DISCUSSION

DR. GRINDON: I think it is pityriasis rubra pilaris, although some of the usual signs are lacking. Ordinarily there are small flat scales, as in dandruff. The outlying lesions, the islands bordering the coast, are usually larger than in this case. I could not make out whether they were connected with the hair follicles. The usual lesions on the dorsal surfaces of the phalanges are indistinct. There is a pithlike accumulation under the free borders of the nails, the palms are much as in pityriasis. What struck me was the picture of the thing as a whole, the sharply outlined patches with fine cross-hatching. The French call it quadrillage, a checkerboard arrangement, the small furrows running across each other at right angles. One spot on the upper part of the abdomen is characteristic.

DR. BROCKELMANN: The condition may be dermatitis mycotica, a form of generalized ringworm infection.

DR. SNIDER: I made smears and cultures and looked for mycelia four different times. The first time I found some in the nail scrapings, but the results of the remaining examinations were negative.

DR. LANE: The diagnosis in this case was pityriasis rubra pilaris, although some of the usual characteristics of that disease are absent. The patient, however, stated that until a strong salve was used the scalp was covered with dry, thick scales. The primary focus has always been follicular, even though at present this feature is not marked. The appearance today closely simulates that in the two cases of pityriasis rubra pilaris presented at the Mississippi Valley Dermatological conference in Chicago last January, one by Dr. Ormsby and the other by Dr. Foerster.

DR. GRINDON: At a meeting in Cincinnati last fall, four cases of pityriasis rubra pilaris were shown. Three in one family — the father, daughter and grandson—were shown together. Although heredity has been mentioned, this is a rare combination.

ANGIOKERATOMA. Presented by DR. W. W. COULTER.

A white man, aged 66, an electrician, was transferred from the City Hospital where he was being treated on the medical division. His father had died of cancer of the throat at the age of 97. His mother had died of "hemorrhage of the bowels." The patient had an appendectomy thirty years previous to presentation. He gave a history of freezing his feet about twenty years ago.

About three months previous to presentation, the patient stepped on a nail; the wound healed, and shortly thereafter the ankles began to swell and crusts appeared. There was no pain or other associated symptoms.

Shortly after admission to this hospital, the patient had a violent chill and the temperature was elevated to 104.4 F. Physical examination showed the following: (1) arteriosclerotic heart disease with auricular fibrillation; (2) streptococcic cellulitis of the right thigh, and (3) paralysis agitans.

The eruption was confined to the feet and legs. The right extremity showed the following distribution: entire dorsal surface of large toe and second toe, and partly of third and fourth toes; anterior surface and furrow of great toe; medial aspect of dorsum of foot; and anterior surface of knee. The eruption presented a dirty grayish appearance, having a keratosic surface forming a sheet on the first and second toes, and scattered discrete keratosic lesions on other areas noted. Some lesions were free from keratosis and appeared to be slightly raised, blue-red tumors the size of a pea. The left extremity showed: several groups of lesions on the big toe, a few on the second toe and scattered lesions along lateral aspect of the dorsum to the back of the heel. These lesions were less keratosic than those on the right foot. There was one blue-red tumor the size of a cherry seed on the knee and several discrete lesions, the size of a pea, on the upper part of the calf.

The blood pressure was 110 systolic and 60 diastolic. The white blood cells numbered 12,400; the red blood cells, 3,760,000, with 60 per cent hemoglobin.

The differential count revealed polymorphonuclears, 82 per cent; lymphocytes, 18 per cent. The Wassermann reaction was negative. The specific gravity of the urine was 1.016; there was no sugar, albumin 2 plus and no casts. The results of biopsy were not reported.

DISCUSSION

DR. SNIDER: I made a diagnosis of angiokeratoma.

DR. LANE: I agree with the diagnosis. Clinically, this is a true case, as is evidenced by the history of frostbite and chilblains followed by the appearance of hemorrhagic spots and papules, which became hyperkeratosic and warty-like. As Wile has recently shown, there is a definite histologic picture in angiokeratoma of hemorrhage from the vessels, epithelial overgrowth, loss of elastic tissue and perivascular inflammation, which is not found in senile angiomas and angioma of the scrotum. Unfortunately, the biopsy in this case is not ready, but will be reported on later.

NEW YORK DERMATOLOGICAL SOCIETY

PAUL E. BECHET, M.D., *Secretary*

Regular Meeting, May 28, 1929

HOWARD FOX, M.D., *Chairman*

PEMPHIGUS FOLIACEOUS. Presented by DR. THRONE.

M. S., a Polish woman, aged 30, married, said that the condition began with a bulla on the right breast two and one-half years before presentation. Similar lesions appeared later on the back, legs and arms. She was admitted to the New York Skin and Cancer Hospital on Oct. 25, 1928. About fourteen months previous to this date the condition had become generalized.

On admission to the hospital, the patient showed a generalized scaly eruption. There was general adenopathy. She was in poor physical condition, and had lost about 30 pounds (13.6 Kg.). Physical examination showed heart sounds of poor quality; otherwise, the heart was normal. The pulse rate was 96. The temperature and lungs were normal. There was a purulent discharge from the left ear.

The patient had had two miscarriages, and one living child who was about 3½ years old. The patient claimed that the skin condition began after the birth of this child, and that since the birth of the child she had not menstruated.

The Wassermann reaction of the blood and spinal fluid and the colloidal gold curve were negative. The differential blood count, except for an eosinophilia of 7 per cent, was negative. The number of white cells varied from 9,600 to 14,000; the red cells numbered 4,390,000; hemoglobin was 80 per cent. On December 1, the urine chlorides were 15.63 per liter; on Feb. 7, 1929, 8.44 per liter. Chemical analysis of the blood was done once a week. The sugar varied from 72.3 to 143.4, the average being between 80 and 90. The chlorides varied from 433.5 to 550; only one examination showed more than 500. The urea nitrogen varied from 6.5 to 16, the uric acid from 2.19 to 5.27.

Chemical analysis of the spinal fluid showed: sugar, 64.4; chlorides, 717.7; urea nitrogen, 7. On January 24, the basal metabolism showed an average of plus 26.1; on February 1, plus 36.6; on February 25, plus 33.8, and on April 16, plus 22.1.

With 0.9 per cent solution of sodium chloride, resorbtion of the wheal was complete in twelve minutes. Controls with this solution showed one and one-half hours.

Arsenic in the form of solution of potassium arsenite was given. The dose was up to 54 mm. per day. In addition, tablets of sodium thiosulphate, 0.5, were given three times a day. In spite of this, the body had become deeply pigmented.

On April 16, the urine chlorides in twenty-four hours were 10.87. The ratio of free acid to ammonia was approximately 1:1. The chloride wheal was resorbed in one hour. On May 8, chemical analysis of the blood showed: chlorides, 520; sugar, 82.9; urea nitrogen, 14.9, and uric acid, 3.59.

The general condition was somewhat improved. A few new bullae appeared from time to time. The patient also received ovarian preparations from time to time, and she had menstruated at infrequent intervals. The reestablishment had had no apparent effect on the course of the disease. There also seemed to be no relationship between the elimination of chlorides and the appearance of the bullae, nor between the resorption time of the chloride wheal and the appearance of new lesions.

On May 15, chemical analysis of the blood showed: chlorides, 429.3; sugar, 80.3; urea nitrogen, 13.1; uric acid, 3.76; calcium, 9.3, and carbon dioxide combining power, 67.9 per cent.

On May 20, the patient had an outbreak of new bullae.

On May 28, the chloride wheal resorption time for 0.4 solution of sodium chloride, was twenty minutes; for 0.9 solution of sodium chloride, thirty minutes.

A Case for Diagnosis (Lupus Erythematosus?) Presented by Dr. Williams for Dr. Lane.

A. B. P., a man, aged 48, said that the trouble had begun fifteen years previously on the chin and cheeks. He had been treated by various physicians, without relief. Among other treatments he had had an ammoniated mercury ointment, and about twenty roentgen treatments once a week.

He presented five distinct affected areas: (1) the chin, around the angles of the mouth to the alae nasi, extending 1 inch (2.5 cm.) beyond the angles of the mouth. The upper borders were absolutely sharp, the lateral and lower borders vague. (2 and 3) In front of each ear was a large irregular patch, about 1½ by 2 inches (3.77 by 5 cm.), the anterior border was sharp, the posterior border vague. (4 and 5) On the sides of the neck under the ears were indefinite, irregular patches, about 3 inches (7.6 cm.) in diameter.

All the lesions were dull red when shown, but sometimes became fiery, and often were scaly in the morning and usually clear in the afternoon. There was neither moisture nor pustules at any times; telangiectasia was slight. There was a slight tendency to atrophy, especially visible at the sharp margins.

DISCUSSION

Dr. Eller: The patient showed telangiectasia and slight scaling. I should think of lupus erythematosus in this case.

Dr. Whitehouse: I think that the condition almost warrants a diagnosis of seborreic eczema, for the patient has had seborrhea all his life; but its fixed character would point to lupus erythematosus without atrophy. It goes to illustrate how apt was Hebra's designation of lupus erythematosus as "seborrhea congestiva."

Dr. Rulison: I think that the case is one of lupus erythematosus.

Dr. Williams: I also thought of lupus erythematosus, on account of the long duration, chronic character, slight scaling and slight tendency to atrophy.

Miliary Tuberculosis of the Face. Presented by Dr. MacKee.

Mrs. A. W., aged 35, was first seen in the Skin Department of the New York Post-Graduate Medical School and Hospital one month before presentation. Scattered over the entire face were several hundred brownish follicular nodules, the size of a pinhead. These tiny nodules were both obtuse and acuminate. They were rather shiny and for the most part smooth, but many of them were capped with either a scale or a crust. Many lesions had undergone spontaneous involution, leaving tiny pits. The duration of the disease was four months. The remainder of

the cutaneous surface was normal. There were no important points in the past history. General, physical and laboratory examinations were negative, including a roentgenographic examination of the chest. One lesion was removed for histologic study. The microscopic diagnosis was tuberculosis. The patient had received four fractional roentgen treatments, and all the lesions were improved.

DISCUSSION

DR. THRONE: This patient came to the Skin and Cancer Hospital some months ago, and we diagnosed the condition as some form of tuberculosis. She was given gold compound and injections of tuberculin, neither of which helped.

DR. WHITEHOUSE: Miliary tuberculosis is my diagnosis.

A CASE FOR DIAGNOSIS (SARCOMA?). Presented by DR. HOWARD FOX.

Mrs. C. L., aged 71, had been previously presented on two occasions (ARCH. DERMAT. & SYPH. **3**:434 [Sept.] 1923, and **11**:885 [June] 1925). Since the last presentation she had had several recurrences of firm, hemispherical, dull red, smooth, nonitchy lesions on the forehead or temple. These had invariably yielded rapidly to unfiltered roentgen rays in comparatively small doses. A recent examination of the blood showed a normal leukocyte and differential count. Histologic examination by Dr. Highman showed a sarcoma with round cells.

DISCUSSION

DR. FRASER: I do not think that this condition is a round cell sarcoma. These growths have been present for fifteen years without disturbance of the patient's health. My impression is that we are dealing here with an inflammatory process. There is some resemblance in the histologic picture to lymphosarcoma, but what is lymphosarcoma? There are those who believe that it is not a neoplasm.

DR. HIGHMAN: Histologically, it seemed to me that the lesion was an aberrant type of round cell sarcoma, or possible a lymphosarcoma. I have the impression that the lesion is a neoplasm rather than an inflammatory condition. It occurred to me that it might be an example of Brocq's mycosis d'emblée. All of Brocq's original cases terminated with the picture of lymphosarcomatosis. The question of whether lymphosarcoma is inflammatory or neoplastic (and some of the pictures suggest inflammation) is not settled, so that with the general question in doubt Dr. Fraser's remarks are sound. I should hesitate between an atypical form of Brocq's mycosis d'emblée and lymphosarcomatosis.

DR. MACKEE: The result of roentgen treatment is of some diagnostic importance. Many of the inflammations and granulomas, particularly the lymphoblastomas, respond amazingly well to fractional roentgen treatment; whereas the neoplasms, with the exception of some cases of lymphosarcoma, do not respond nearly so rapidly as a rule. Some cases of lymphosarcoma are exceedingly "radiosensitive," but never so much so as is encountered in the majority of cases of mycosis fungoides. The fact that these lesions responded so well to roentgen treatment supports to some extent Dr. Fraser's impression that the lesion is a granuloma or inflammation rather than a neoplastic condition.

DR. WHITEHOUSE: From the long duration of fifteen years, I throw out the idea of its being a malignant process. Dr. Fraser's description of lymphosarcoma would place it in the category of an inflammatory process rather than that of a malignant neoplasm, and what Dr. MacKee said about the influence of the roentgen ray would tend to that classification.

DR. HOWARD FOX: On account of the long duration I have hesitated to call this condition sarcoma. My first diagnosis was a possible sarcoid. As Dr. MacKee has said, conditions of this kind respond remarkably well to the roentgen rays.

DR. MACKEE: I have seen lymphosarcoma disappear with amazing rapidity under roentgen therapy, but I have never known such a lesion to respond so

rapidly, so favorably and as a result of such a small dose as in the case of previously untreated mycosis fungoides.

DERMATITIS FACTITIA. Presented by DR. HOWARD FOX.

Mrs. L. H., aged 27, born in the United States, had suffered from an eruption of the cheek for four months. She presented a group of a dozen lesions the size of a pinhead on the right cheek, grouped in a circle the size of a quarter. The lesions were dull red and disappeared only partly under glass pressure. There was no ulceration, crusting or evidence of scratching. The lesions were somewhat tender. They presented an unnatural appearance, which did not correspond to any recognized dermatosis. There was no evidence of acne or other cutaneous dermatosis. The patient was distinctly of the neurotic type. There was an absence of corneal and pharyngeal reflexes.

DISCUSSION

DR. TRAUB: I think that the condition is traumatic, the primary lesion being a comedone which the patient has attempted to remove. It happened that the lesions occurred in a circular form, hence the peculiar configuration.

DR. WHITEHOUSE: I believe that the lesions are self-produced.

ALOPECIA UNIVERSALIS AND KERATOSIS PILARIS. Presented by DR. MACKEE.

J. C., a boy, aged 10, from the Department of Dermatology of the New York Post-Graduate Medical School and Hospital, had had several attacks of complete alopecia of the scalp and had always made a good recovery. A few months before presentation all the hair disappeared from the scalp, eyebrows and eyelids. When presented there was no hair on the body, not even lanugo hair. Dr. Timme had found a low blood calcium, and had prescribed calcium and iodine. The general physical examination was negative. Six months previous to presentation, patches of shiny flat follicular papules developed on the abdomen. There were eight patches, and they varied in size from that of a dime to that of a palm. This eruption had been diagnosed as lichen scrofulosorum. It was presented as an example of keratosis pilaris.

A CASE FOR DIAGNOSIS (GRANULOMA ANNULARE?) Presented by DR. BECHET.

The patient presented lesions on the backs of the hands which had been present for eighteen months. They were quite large and serpiginous, but to some extent lacked the rolled, waxy border so common in granuloma annulare. The center of the lesions was composed of normal skin. There were no subjective symptoms.

DISCUSSION

DR. WISE: I should suggest the diagnosis of porokeratosis of Mibelli.

DR. TRAUB: I agree with Dr. Wise. The thought that this might be a case of porokeratosis also came to me.

DR. THRONE: I should suggest the possibility of tuberculosis cutis.

DR. MACKEE: The eruption is essentially nodular. The nodules have a waxy appearance and some of them are umbilicated. This evolution with central involution is often encountered in cases of granuloma annulare. Also atrophy is sometimes seen in this disease. While other diseases should be considered, I think that it is a case of granuloma annulare.

DR. ELLER: I incline to the diagnosis of granuloma annulare.

DR. WHITEHOUSE: I should rely on a biopsy for differentiation. The condition has not the earmarks of tuberculosis verrucosa cutis, but does have a warty character in some places which suggests it.

DR. HIGHMAN: I believe that the condition is granuloma annulare. I did not examine the patient closely, but it does not suggest porokeratosis. As for tuber-

culosis verrucosa cutis, I have never seen so many lesions in such cases. If it is not granuloma annulare it is a closely related condition, perhaps sarcoid.

DR. BECHET: The patient came in only today. Dr. Wise's suggestion of porokeratosis will be taken into consideration, but the clinical evidence is more in favor of granuloma annulare.

DR. HOWARD FOX: I agree with those who consider the condition granuloma annulare. That is a fairly common disease, whereas porokeratosis is exceedingly rare. The latter is so rare that none of us have probably seen more than one case in our entire experience.

PERIADENITIS MUCOSA CHRONICA RECURRENS. Presented by DR HOWARD FOX.

J. J. O., a man, aged 34, born in the United States, a clerk, had suffered from recurring sores of the lips for fifteen months. Previous to the attack of three months' duration, for which he was presented, the disease had suggested" canker sores," lasting on an average of three weeks, disappearing spontaneously and followed after a week or so by new lesions. They occurred singly on an average of about once a month. They were very sore to the touch and when the patient ate. The eruption was always confined to the mucous surface of the lips. The last attack had been severe and consisted of two deep ulcerations which were extremely tender and interfered with sleep. The patient lost 15 pounds (6.8 Kg.), which he ascribed to difficulty in eating. He gave no history of syphilis or tuberculosis.

Examination showed a horseshoe shaped ulceration about 1 inch long on the mucous surface of the right side of the lower lip. It was covered with a thick yellowish membrane and was sharply demarcated, extremely tender and infiltrated. There was considerable inflammatory reaction at the border. A second similar lesion was present on the left side of the upper lip. Several small, firm, submaxillary glands could be palpated. The patient had been previously treated at the Cornell Clinic, where examination for Vincent's organisms was said to be negative, and where six injections of neoarsphenamine were given without benefit. The Wassermann reaction was negative, and no tubercle bacilli were found in smears. The teeth were in good condition. The patient did not use tobacco or alcohol. The condition had recently improved under treatment with the roentgen ray.

DISCUSSION

DR. FRASER: I was unable to make a diagnosis.

DR. RULISON: I could not make a definite diagnosis.

DR. THRONE: I was under the impression that in periadenitis the lesions are usually single, and had no idea that they occurred in such large numbers. As I remember, the patients always complained of a central plug which had to be removed before they got relief.

DR. MACKEE: My recollection and conception of periadenitis mucosa necrotica recurrens are that the lesion begins as a nodule which breaks down into an ulcer. The clinical picture is suggestive of papulonecrotic tuberculid. The late Dr. Fordyce and others who have studied this disease clinically and histologically considered it to be identical with papulonecrotic tuberculid. The lesions are usually multiple and disappear spontaneously, and the disease is characterized by remissions and exacerbations.

In the last two years I have had six or eight patients with an eruption exactly like that of the patient presented by Dr. Fox, and in each instance the eruption has been exactly the same. The multiple lesions and the remissions and exacerbations suggest periadenitis mucosa necrotica recurrens, but there are never any nodules and the lesions are exceedingly painful. Drug eruptions, Vincent's angina, dermatitis venenata, pemphigus and leukemia have been ruled out in these cases so far as it is possible to do so. I have not included these cases under the caption of periadenitis because the lesions are never nodular and because they are so

painful. I have always called them, for the want of a better diagnosis, multiple recurring apthous ulcers, although, of course, they may represent cases of aberrant periadenitis.

DR. RULISON: The ulceration has been deep and tender, and pain is severe. The ulcers have really been very deep and are healing with a great deal of scarring. I noticed that where they had healed there is considerable puckering, which I think is due to contraction of the scar. I have never seen any nodules.

DR. HIGHMAN: While not offering the diagnosis with any conviction, I think that it is conceivable that we ought to consider the prodromal period of pemphigus. In many instances pemphigus has a history of a year or more of initial involvement of the mouth. While I do not think that it is so in this instance, the possibility should not be ignored.

DR. WISE: Provided the tubercle bacillus is not found in the lesions, I should be inclined to agree with the diagnosis. The condition is similar, if not identical, to that which Sutton described in his textbook.

DR. HOWARD FOX: I am not at all sure about this rare condition, as I am not any more familiar with it than any of those present. It is more by exclusion than anything else that I decided to fasten this name on the eruption. Dr. Highman's suggestion of pemphigus I did not consider, despite the fact that the patient had lost a good deal of weight. He has had great difficulty in eating, and the loss of weight may have been due to that. The lesions we see in pemphigus are superficial bullae which rupture quickly and leave an excoriated surface, rather than a deep ulceration as seen in this case. The patient is entirely willing to cooperate, and to have a biopsy which will be reported on later.

PYODERMA. Presented by DR. MACKEE.

A. F., a fruit salesman, aged 45, about eight months before presentation developed what was supposed to be a carbuncle on the back of the neck. This was incised, and several ounces of pus was evacuated. Since then there had been a constant development of large and small deep-seated pus pockets over the back of the neck, with the development of widespread vegetations and considerable scarring, much of which was caused by numerous surgical operations.

A few months before presentation a general pathologist made a diagnosis of actinomycosis. The patient entered the dermatologic department of the New York Post-Graduate Medical School and Hospital about two months before presentation. The general physical examination, chemical analysis of the blood, Wassermann test of the blood, urinalysis, blood count and roentgenographic examinations gave negative results. Dr. Satenstein made a careful histologic examination but found nothing but inflammation. Dr. George M. Lewis had repeatedly searched for fungi in the pus, tissue and hairs, but none had been found. Inoculation experiments in animals had not been performed. The condition was slowly improving under ultraviolet irradiation, wet dressings, multiple incisions, rest, nutritious diet and a few fractional roentgen treatments given when the patient first came to the clinic.

When presented the skin of the back of the neck was exceedingly thick but not keloidal. This thick tissue was irregular—lobulated—with here and there a sinus discharging a little pus. Diagnoses such as dermatitis papillaris capillitii, fungous diseases, perifolliculitis capitis absedens et suffodiens had been suggested, but the case was presented simply as pyoderma.

DISCUSSION

DR. WHITEHOUSE: I agree with Dr. MacKee that the condition is pyoderma. I do not think that it is dermatitis papillaris capillitii.

DR. HIGHMAN: I do not see that anything is gained by giving suppurative diseases of the skin all sorts of different names; therefore, I agree with the diagnosis of pyoderma.

DR. WILLIAMS: The condition belongs to that large group of inflammatory conditions occurring particularly on the scalp, neck and eyebrows, and involving the

hair follicles and sebaceous glands, in which the immediate reaction may be rather severe, with suppuration, or so mild that there is scarcely a visible redness, but which usually go on to atrophy or scarring.

DR. RULISON: The diagnosis of carbuncle was probably correct, but the surgical treatment was not thorough enough to clear up the infection.

PAGET'S DISEASE. Presented by DR. MACKEE.

Mrs. R. D., aged 48, from the Dermatological Department of the New York Post-Graduate Medical School and Hospital, was presented for an eruption which had first appeared two years before presentation. It had spread very slowly. It had never disappeared and had never really improved. When presented the patient exhibited an eczematous-like eruption of the right breast, surrounding the nipple. The patch extended about two-thirds around the nipple and it did not extend more than 1 inch from the nipple. The nipple itself was involved and perhaps slightly retracted. When presented the eruption was dull red, smooth and dry. The patient said, however, that there had been exudation and exfoliation at times. There were no subjective symptoms. A biopsy had been performed by Dr. Satenstein and the specimens which were presented to the Society showed typical Paget's disease of the skin and early carcinoma of the breast. Amputation of the breast was to be performed immediately.

DISCUSSION

DR. FRASER: I agree with the diagnosis of Paget's disease. The histologic picture is typical of the Paget lesion, which is now almost universally admitted to be cancer and not a form of eczema as Paget thought, or a precancerous lesion. In addition to the intra-epidermal lesion (Paget's disease), the slides in this case show involvement of the intralobular portions of the mammary ducts. Tumor cells can be seen breaking through these ducts and forming a scirrhous cancer in the tissues of the breast. Paget's disease may be regarded as cancer of the intra-epidermal endings of the lactiferous ducts and should be treated like any other form of cancer. The minimum rational treatment should be radical mastectomy.

DR. ELLER: The more reports I read in the literature the more I feel that when Paget's cells are in the epidermis there is a cancer in every case in the upper part of the milk ducts, and no matter how brief the disease, a mastectomy ought to be done. Recently we had a case of Paget's disease of the nipple, of only one month's duration. A mastectomy was done, and microscopic studies of serial sections of the breast revealed a carcinoma of the upper parts of the milk ducts.

DR. MACKEE: I agree with Dr. Fraser and Dr. Eller that in all cases in which a diagnosis of Paget's disease is microscopically verified, amputation should be performed immediately. In a given case we may find the Paget cell in the epidermis and the ducts may appear to be normal. Such an observation may be misleading because in many such instances, when the entire breast is removed and sectioned serially, deep carcinoma is found. I, therefore, favor radical excision if the condition is in an operable case; but if the patient is very old or in bad condition from some other disease and the expectancy of life is not great, I should not hesitate to use intensive filtered roentgen treatment in preference to surgical intervention.

TATTOOING ON THE FACE. Presented by DR. KINGSBURY.

An Armenian woman, aged 22, whose parents were killed during the early part of the World War, was captured by the Kurds when about 7 years of age. Later she was transferred to a Turkish harem. While there she was tattooed on the temples, chin and neck. The more conspicuous and significant designs on the chin had been partially removed. This was said to have been done by the use of crystals of citric acid, the application of which was made by the patient herself.

DISCUSSION

DR. HOWARD FOX: I have a photograph of an Armenian girl who was tattooed in a similar manner by the Turks. This patient was previously presented (ARCH.

DERMAT & SYPH. **2**:255 [May] 1920). We have had some experience in our clinic with removing tattoo marks according to the method recently described by Shie. The lesion is tattooed with the electric tattoo needle, a 50 per cent aqueous solution of tannic acid being dropped on the skin during the procedure. The surface is then painted with a silver nitrate stick, forming a slough which falls in a few weeks leaving a fairly good scar.

SCLERODERMA WITH ULCERS AND CALCAREOUS DEPOSITS. Presented by DR. HOWARD FOX.

D. G., a man, aged 38, Jewish, had had gangrene of the great toe eight years previously. This followed strong applications to a bunion, and as a result amputation of the great toe was performed. Four years later he noticed a tightness of the skin of both feet. Ulcers developed soon after. Previous treatment included the use of salves, baking the feet and one exposure to the roentgen rays.

Examination showed well marked hidebound skin of the feet with some atrophy. On the lower third of the anterior surface of the right leg and over the right external malleolus were deep, well defined ulcerations 1½ inches (3.77 cm.) in diameter, with serosanguinolent discharge and sloughing base. A similar ulcer was present above the left heel. Sensations of heat, cold and pain were increased in the foot. There was ankylosis of the ankles and toes, the left great toe being absent as a result of former amputation. The patient walked with a stiff stepping gait and with a twisting of the hips. His general appearance was that of a man much older than 38. He was of small stature and had a scanty growth of hair, especially of the eyebrows and to a less extent of the beard. There was well advanced Hippocratic baldness. The pubic hair was fairly normal and of masculine type. The skin, except on the feet, was normal and showed no pigmentation. He perspired as freely as the average person. The eyeballs were somewhat prominent; there were marked myopia and double cataract. The teeth were apparently in excellent condition, two being absent. His hearing was good. The hands were small, the fingers short and the nails normal. The genitals were of normal size. There was no history of Raynaud's disease. The radial pulse was normal, and the pulsation in the dorsalis pedis artery was barely perceptible. The Wassermann reaction was negative. Histologic examination of tissue at the edge of one of the ulcers showed typical changes of scleroderma. A roentgenogram of the foot was made by Dr. L. T. Le Wald, who reported as follows:

"There was evidence of osteoporosis of the bones of both feet. The right foot showed ankylosis between the metatarsal and first phalanx of the great toe. The arteries showed an extreme degree of calcification extending up to the middle of the leg on each side. There was a peculiar deposit of lime salts along the posterior and internal border of the fibula on each side, together with some bony deposit about the external border of the left tibia. These peculiar bone deposits were unusual and were also associated with some calcification about the tendon Achilles at its insertion."

DISCUSSION

DR. BECHET: I think that the patient should have a thorough endocrinologic examination.

DR. ELLER: Such a condition of the bones may be found in leprosy, syphilis and scleroderma. Any of these conditions may cause rarefaction of the bones and ankylosis.

DR. WILLIAMS: I think that it is an ordinary case of scleroderma, a disease which is often accompanied by just such chronic ulceration, osteoporosis, ankylosis and deposits of calcium in various tissues, such as the tendon Achilles. I have here two lantern slides made from roentgenograms of a case which I reported several years ago. One shows a foot in which the disease was not very far advanced, and the bones were approximately normal. The other foot was very badly diseased, ulcerated and had been useless for several years; the bones of this foot were porous, the outlines indistinct and there were patches of calcification around the os calcis and extending up the tendon Achilles.

DR. HIGHMAN: I agree with what Dr. Williams has said. Calcification is not rare in scleroderma. The pressure of the hidebound skin on the hard tissues produces the changes which may be observed here; finally the microscopic appearance of the lesion is that of scleroderma. In the final analysis, perhaps scleroderma is an endocrine disturbance. Johnston thought that it was a disturbance of the pituitary gland, and there was some evidence to sustain his point of view. I begin to take the observations of endocrinologists seriously when they do not prescribe thyroid substance or compound solution of cresol. I think that this case will remain a case of scleroderma.

DR. WISE: I agree with the diagnosis of scleroderma involving the bones.

DR. THRONE: We have found a high plus basal metabolic rate in many similar cases.

DR. MALONEY: When I first saw this patient about a week ago I made a diagnosis of scleroderma, and I see no reason for changing it now. The roentgen report was a surprise to me as I did not know that the bone changes here shown were ever present in this disease. Dr. Williams has just demonstrated these changes very well in the slides of his case of scleroderma.

DR. HOWARD FOX: I am glad to have this discussion of the case, and will try to study it from the endocrine standpoint. When I first saw the patient the disease did not impress me as scleroderma. It is not the usual progressive type, beginning in the hands and extending up the arm and including the face, and later on other parts. I am indebted to Dr. Williams for reminding me of the case he described in detail, which I had forgotten.

A CASE FOR DIAGNOSIS (LICHEN PLANUS?). Presented by DR. MACKEE.

Mr. S., aged 35, from the Dermatological Department of the New York Post-Graduate Medical School and Hospital, exhibited an eruption on the buttocks of the external and posterior aspects of the left thigh of seven years' duration. The patient said that the eruption began on the left buttock and very slowly spread. The eruption was violaceous and consisted of erythematous, slightly infiltrated, small, scaly, irregular patches varying in size from that of a dime to that of a quarter. Interspersed throughout the entire area were numerous discrete and grouped papules. There were numerous scratch marks, and the patient complained of itching. Various diagnoses had been considered in the several clinics consulted by the patient—lupus vulgaris erythematoides, lichen planus, mycosis fungoides, tinea, etc. Treatment with a gold compound had been administered, also sodium thiosulphate and many topical remedies, without benefit. Several years previous to presentation the patient had received a course of fractional roentgen treatment which caused temporary improvement. The patient had entered the Post Graduate clinic only a few days before presentation. Investigation had been started immediately but the laboratory data were not yet available.

DISCUSSION

DR. WILLIAMS: Despite the sharp outline of the border on each thigh near the scrotum, I do not think that the condition is tinea. The borders are too irregular, are not inflammatory enough and there is slight atrophy in one or two places. I do not think that it is tinea of any kind.

DR. WHITEHOUSE: We see many cases of dermatitis herpetiformis now located on the buttocks, and we see cases like that existing for a long time. I think that dermatitis herpetiformis should be considered in making the diagnosis.

DR. HIGHMAN: Two contradictory features struck me: the large lesions on the hip, not lichenified, but indicating the presence of a chronic edema, which suggested early mycosis fungoides, and the lesions on the buttocks, fading, pigmented and centrally atrophied, lesions that are peripherally edematous and firm. These suggested morphea. Why the itching, I do not know. These two ideas suggest themselves to me, and I offer them for what they are worth in studying the case.

DR. HOWARD FOX: If the patient had an extensive eruption of the same character, I should certainly think of dermatitis herpetiformis.

PAPULONECROTIC TUBERCULID SUCCESSFULLY TREATED WITH SULPHARSPHENAMINE. Presented by DR. HOWARD FOX.

Mrs. J. V., aged 46, born in the United States, was first seen two years before presentation, when she exhibited a papulonecrotic tuberculid of four years' duration. At that time the eruption was profuse and situated chiefly on the extremities and to a less extent on the trunk. The eruption was symmetrical and had left numerous characteristic pitted scars. The Wassermann test had been made on four occasions, although there was no history of syphilis, and the reaction was always negative. The patient was advised to try the effect of injections of salicylate of mercury. Thirty-six injections were given by her physician without benefit. Later she was advised to have small doses of neoarsphenamine. She received, however, thirty-six injections of sulpharsphenamine intramuscularly, usually at weekly intervals, including nine injections of 0.3 Gm., a similar number of 0.4 Gm. and eighteen of 0.6 Gm. The eruption, according to her statement, began to improve after the sixth injection, and at the end of the course of treatment had entirely disappeared. She remained well for six months, but a fresh lesion appeared two weeks before presentation. During the treatment, the patient also gained 15 pounds and her general health improved greatly.

DISCUSSION

DR. WISE: I have had temporary cures of papulonecrotic tuberculid following injections of neoarsphenamine.

SARCOID OF BOTH THE DARIER-ROUSSY AND THE BOECK TYPE. Presented by DR. BECHET.

S. K., a man, aged 52, said that he first noticed a small tumor on his right hand nine months previously. One or two months later, other tumors appeared on the arms and trunk, and at the time of his appearance at the clinic he presented several infiltrated, indurated, violaceous, red plaques, and from rounded to oval, various sized subcutaneous and cutaneous tumors, from a dull red to yellowish. These tumors and plaques were painless on palpation.

Directory of Dermatologic Societies *

NATIONAL DERMATOLOGIC SOCIETIES

American Medical Association, Scientific Assembly, Section on Dermatology and Syphilology

William H. Guy, Chairman, Jenkins Arcade, Pittsburgh.
Francis E. Senear, Secretary, 55 E. Washington St., Chicago.
Place: Detroit. Time: June 23-27, 1930.

American Dermatological Association

Udo J. Wile, President, University Hospital, Ann Arbor, Mich.
C. Guy Lane, Secretary, 416 Marlborough St., Boston.
Place: Cleveland. Time: 1930.

British Association of Dermatology and Syphilology (Canadian Branch)

Gustave Archambault, President, 544 East Sherbrooke St., Montreal.
Albéric Marin, Secretary, 544 East Sherbrooke St., Montreal.

LOCAL DERMATOLOGIC SOCIETIES

Los Angeles County Medical Association, Dermatologic Section

George F. Koetter, Chairman, 1136 W. 6th St., Los Angeles.
H. Sutherland-Campbell, Secretary, 1936 Wilshire Blvd., Los Angeles.

San Francisco Dermatological Society

Thomas Clarke, Madison-Lake Apts., Oakland, Calif.
H. J. Templeton, Secretary, 3115 Webster St., Oakland, Calif.

Atlanta Dermatological Society

Howard Hailey, President, Candler Bldg., Atlanta, Ga.
H. S. Alden, Secretary, Medical Arts Bldg., Atlanta, Ga.

Chicago Dermatological Society

Harry R. Foerster, President, Colby-Abbott Bldg., Milwaukee.
E. A. Oliver, Secretary, 55 E. Washington St., Chicago.

Iowa and Western Illinois Dermatological Association

Robert E. Jameson, President, 1015 First National Bank Bldg., Davenport, Iowa.
A. T. Leipold, Secretary, 1419½ 5th Ave., Moline, Ill.

Louisiana Dermatological Society

M. T. Van Studdiford, Pere Marquette Bldg., New Orleans.
Thomas A. Maxwell, Secretary-Treasurer, 1119 Union Indemnity Bldg., New Orleans.

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* Secretaries of dermatologic societies are requested to furnish the information necessary for the editor to make this list complete and to keep it up to date.

BALTIMORE AND WASHINGTON DERMATOLOGICAL SOCIETY

Frank J. Eichenlaub, President, 1835 Eye St., N. W., Washington, D. C.
Leon Ginsburg, Secretary, 1800 N. Charles St., Baltimore.

DETROIT DERMATOLOGICAL SOCIETY

Arthur E. Schiller, President, 10 Peterboro St., Detroit.
William G. Schlegelmilch, Secretary-Treasurer, 7769 Grand River, Detroit.

MINNESOTA DERMATOLOGICAL SOCIETY

Edward C. Gager, President, 350 St. Peter St., St. Paul.
D. D. Turnacliff, Secretary, Yeates Bldg., Minneapolis.

ST. LOUIS DERMATOLOGICAL SOCIETY

Robert H. Davis, President, Missouri Bldg., St. Louis.
Norman Tobias, Secretary, Missouri Bldg., St. Louis.

NEW ENGLAND DERMATOLOGICAL SOCIETY

Walter T. Garfield, President, 19 Bay State Rd., Boston.
Rudolph Jacoby, Secretary, 270 Commonwealth Ave., Boston.

BRONX DERMATOLOGICAL SOCIETY

George Spinner, Chairman, 811 Cauldwell Ave., Bronx, N. Y.
Ralph Breakstone, Secretary-Treasurer, 783 Beck St., New York.

BROOKLYN DERMATOLOGICAL SOCIETY

E. Almore Gauvain, President, 47 Halsey St., Brooklyn.
A. M. Persky, Secretary, 1750 Ocean Pkwy., Brooklyn.

MANHATTAN DERMATOLOGICAL SOCIETY

David L. Satenstein, Chairman, 57 W. 57th St., New York.
Mihran B. Parounagian, Secretary, 126 E. 39th St., New York.

NEW YORK ACADEMY OF MEDICINE, SECTION OF DERMATOLOGY AND SYPHILIS

John Frank Fraser, Chairman, 100 W. 59th St., New York.
Max Scheer, Secretary, 3 E. 53d St., New York.

NEW YORK DERMATOLOGICAL SOCIETY

Paul E. Bechet, President, 30 E. 40th St., New York.
J. Frank Fraser, Secretary, 100 W. 59th St., New York.

MEDICAL SOCIETY OF THE STATE OF NEW YORK, SECTION ON DERMATOLOGY AND SYPHILOLOGY

Walter J. Highman, Chairman, 853 7th Ave., New York.
Albert R. McFarland, Secretary, 348 University Ave., Rochester.

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